VOLUME LXV JUNE, 1955 NUMBER 6

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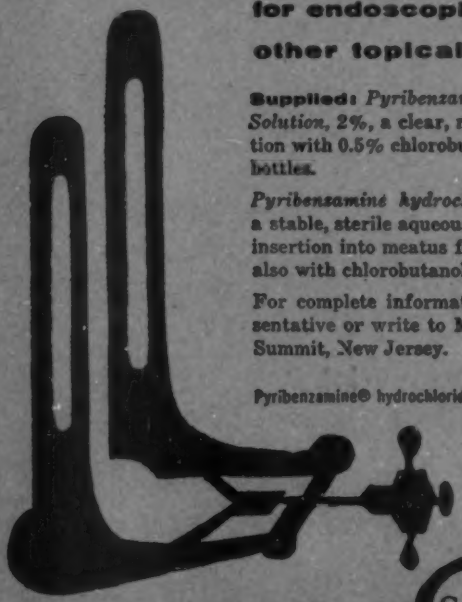

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THE
LARYNGOSCOPE.

VOL. LXV JUNE, 1955. No. 6

LATERAL VESTIGIAL CYSTS AND FISTULAS OF THE NECK.*

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In presenting a subject on the anomalies of the lateral neck one is obliged to review briefly the intricate embryology of the region.

DEVELOPMENT OF THE PHARYNX AND NECK.

A large part of the human neck is formed from the embryonic visceral arches. During the fifth embryonic week the visceral (branchial) arches are seen in the region of the neck and pharynx. Four arches are seen superficially; the fifth and sixth arches are submerged. By the beginning of the seventh week all external traces of the visceral arches have disappeared.

The entodermal lining of the primitive pharynx covers the inner surface of the arches and passes outward in the recesses between them (pharyngeal pouches) to come into contact for a short time with the epithelial covering of the body (ectoderm) which dips in to meet it (visceral grooves or clefts). The membrane formed by the union of the ectoderm and endoderm in the recesses between the arches is called the cleft membrane (closing membrane). It is never ruptured but is soon invaded by the mesoderm of the neighboring arches (see Fig. 1).

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* Presented as Candidate's Thesis at the Fifty-Ninth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Hollywood, Fla., March 16, 1955.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, March 22, 1955.

In each arch there develops a cartilaginous skeleton, an aortic or vascular arch connecting the ventral and dorsal aortas, a large nerve along its anterior border, a smaller one along its posterior, and a muscle component.

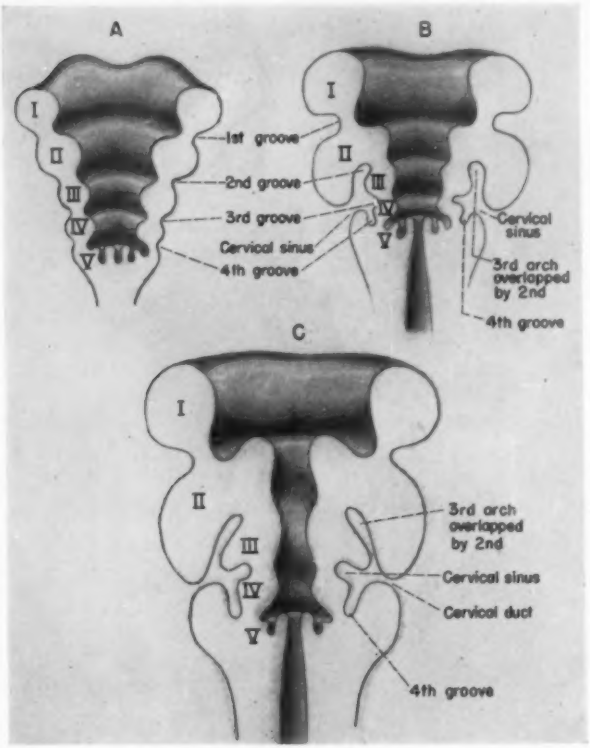

Fig. 1. Showing Early Development of the Pharyngeal Wall.

A. Showing proximity of ectodermal floor of branchial clefts to the entodermal pharyngeal pouches.

B. Rapid growth of the hyoid arch II overshadows postoral arches forming the cervical sinus.

C. Occlusion of the cervical sinus (note its position lateral to groove and pouch III, and its communication with grooves II and IV).

Redrawn from Ward and Hendricks: "Tumors of the Head and Neck."[18]

NERVES OF THE VISCERAL ARCHES.

Each nerve of a visceral arch supplies the muscles of that arch and the pharyngeal pouch and groove lining in front of the arch (see Fig. 3). The Vth nerve and the Gasserian ganglion lie at the base of the mandibular arch. The ganglia of the VIIth and VIIIth nerves are situated at the base of the hyoid arch in front of the otic vesicle. The glossopharyngeal and its ganglia lie behind the otic vesicle at the dorsal wall of the pericardium. The large ganglionic mass of

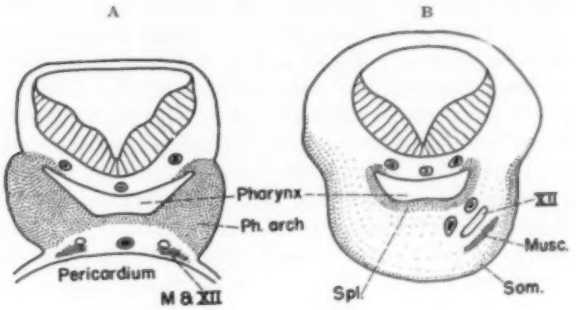

Fig. 2. Showing in the first diagram, the solid condensations of the pharyngeal arches laterally, with loose subpharyngeal mesoderm below, in which lie the vessels and structures (m, XII) of the epipericardial ridge. In the second diagram the condensed mesoderm has been "deposited" on the pharyngeal wall (Spl.), on the one hand, and against the ectoderm (Som.) on the other, so that the XIIth nerve and its muscles lie in loose mesoderm between these two, superficial to the pharyngeal structures and deep to the somatic.

Redrawn from Frazer: "Manual of Embryology."[2]

the vagus lies over the bases of the fourth, fifth and sixth arches and near the dorsal wall of the pericardium. The spinal accessory nerve is a backward extension of the vagus to the cervical region of the spinal cord. Its spinal fibers supply the sternomastoid and trapezius muscles, both of which are of branchial origin. The hypoglossal nerve is not related to the branchial apparatus. The tongue musculature is derived from the last three occipital segments and is somatic in nature (see Fig. 4).

MESODERMAL STRUCTURES.

The lateral pharyngeal walls are made up of concentrated mesoderm in the form of arches. Arches III to VI do not pass to the midline, leaving the central region without an arch concentration (see Figs. 2, 5). The floor of the central region has a limited subentodermal condensation of mesoderm, followed by a loose mesoderm and finally the pericardium. In the lateral wall the entire space between ectoderm and entoderm is filled with dense mesoderm. The arteries extending from the pericardium pass through the loose subpharyngeal layer. During the fifth week the dense mesoderm in the arch divides into a visceral and a superficial condensation with a loose intervening mesoderm (see Fig. 2-B). This change in the arch is associated with disappearance of the arch form on the surface and in the interior of the pharynx. rotate as the pharynx grows forward leaving only their The aortic arches now come to lie on the pharyngeal wall and branches of distribution in the condensations of the wall. The nerves of the arches remain deep to the arteries and in the pharyngeal wall.

The hypoglossal nerve with its muscle cells extends forward between the superficial and deep mesodermal layers. It passes forward superficial to the carotids, thyroid, thymus, larynx, superior laryngeal and glossopharyngeal nerves, and the hyoid. If any connection were to persist between the two walls the nerve would come up against it. None exists, however, until it reaches a branch given off by the external carotid (a visceral artery) to the muscles of the outer wall (sternomastoid branch of the occipital).

The pharyngeal wall and the dorsal structures grow rapidly causing a forward elongation. The relatively slower ventral growth keeps the pharynx curved at first around the top of the pericardium; but as this ventral growth becomes more manifest the separation from the pericardium occurs, and the neck becomes evident.[2]

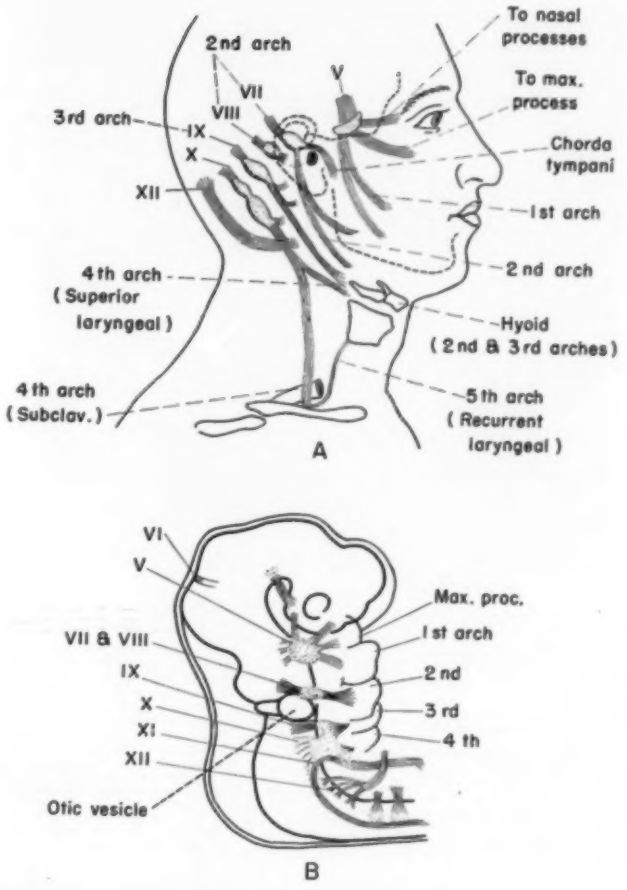

Fig. 3. A—Showing what becomes of the Nerves of the Visceral Arches.

B. The Visceral Arches and Their Nerves and Ganglia in a Human Embryo of the fifth week.

Redrawn from Keith: "Human Embryology and Morphology."[7]

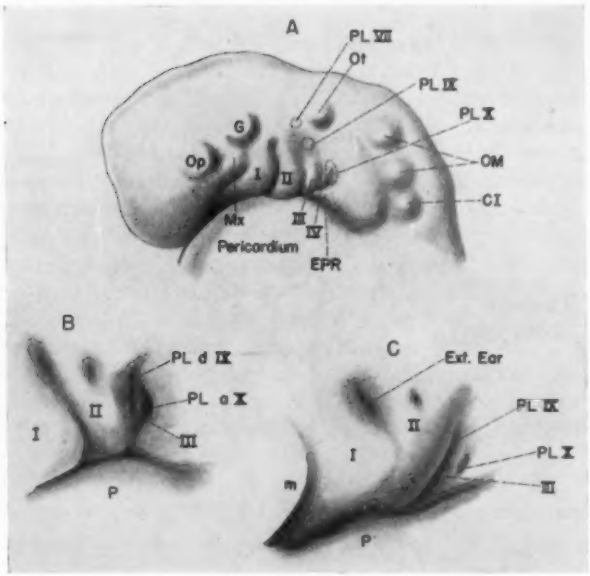

Fig. 4. A—The beginning of the cervical sinus. Neck of a human embryo at the end of the fifth week of development, showing the triangular field in which the third and fourth arches are subsiding. I, mandibular arch; II, hyoid arch; PL VII, placode of facial nerve; PL IX placode of glossopharyngeal nerve; PL X, placode of vagus; CI, first cervical somite; OM, occipital myotomes; Ot, otocyst; G, Gasserian ganglion; Op, slight prominence of eye; Mx, maxillary process; EpR epicardial ridge continual with the caudal boundary of the arch-field. Arches III and IV are being overshadowed by the rapidly growing hyoid arch.

B. View of arches and grooves in a 10 mm. embryo (about five weeks) showing the beginning of the cervical sinus disappearance. The encroachment of the marginal folds narrows the opening into the depressed area; the smaller fourth arch is being covered. P, pericardium; I, II, III, pharyngeal arches. The widely open placodal recess of vagus, PL. a X, is below and behind the narrow duct, PL d. IX, leading to the glossopharyngeal ganglion. The epipericardial ridge is very prominent below and behind the third arch, forming a posterior boundary to the arch area.

C. Arch area in a 12 mm. embryo (about six weeks). Further closure has left only an area of the flattened third arch exposed. The placodal areas are buried; so is the fourth arch. Shows the second arch fused below with the first, but forming the "flexure line" of the neck by its posterior border, behind this third arch, a small area; PL X indicates the point of attachment of the remnant of the tract leading to the placodal vesicle of the vagus, and PL IX points to what is apparently a similar, but less marked track to the placodal vesicle of the glossopharyngeal. P, pericardium; m, mouth.

Redrawn from Frazer.[8]

ECTODERMAL ASPECT AND THE CERVICAL SINUS.

The first (mandibular) arch especially, and also the second (hyoid) grow much more rapidly than the remaining arches. The second arch grows over, reaches the thoracic body wall and buries the third and fourth arches so that an ectodermal space (cervical sinus) becomes enclosed (see Fig. 1). The enclosed cervical sinus is in contact with the second, third and fourth pharyngeal pouches. The cervical sinus is formed in the sixth and seventh weeks of embryonic life but disappears before the end of the second month.

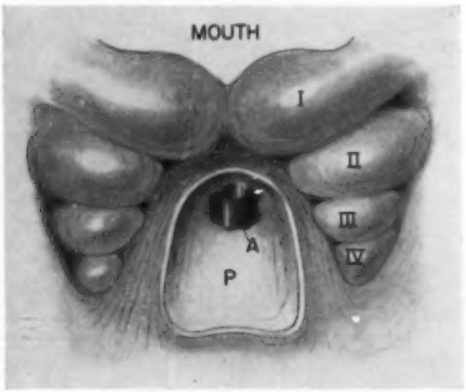

Fig. 5. Somewhat Simplified View from Below of Structures forming Floor of Pharynx, the Pericardium being removed except where it is attached to this floor (P), to show the Structures of the Epipericardial Ridge (interrupted lines) passing Forward to the Mandibular Arch.

A. Aortic stem piercing pericardial roof.

Redrawn from Frazer: "Manual of Embryology."[2]

Frazer showed that the cervical sinus is not completely enclosed, but rather that the third and fourth arches form the floor of a small depressed triangular field (see Fig. 4-A). The anterior border is formed by the hyoid arch, the posterior border is a thick ridge produced by downgrowths from the occipital myotomes. The posterior border extends from behind and below the otocyst, down and back, and then becomes con-

tinuous at an acute angle with a smaller ridge which runs forward above the pericardium (epipericardial ridge). The epipericardial ridge extends medial and forward, so that it is lost to view between the third arch and the pericardium. It ultimately reaches the mandibular arch (see Fig. 5). The epipericardial ridge carries muscle cells from the occipital (and probably upper cervical) myotomes to form the muscles of the tongue, infrahyoid muscles and at least part of the sternomastoid. The hypoglossal nerve arising near the occipital myotomes grows forward with these muscle cells to reach the mandibular arch.[2]

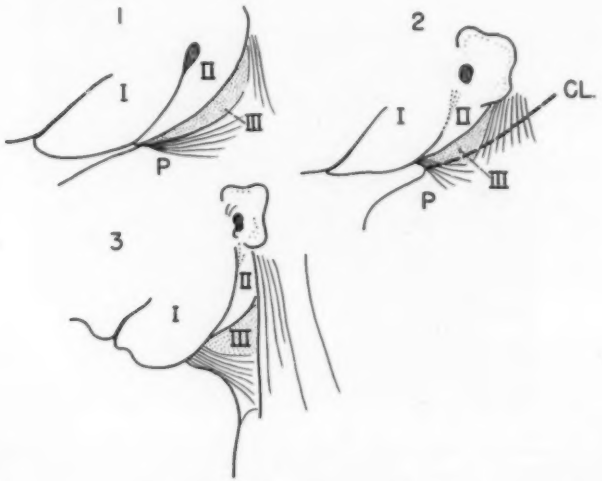

Fig. 6. Three schemes illustrating formation of ventral part of neck. The first shows the first three arches in position, as at 12 mm., with the muscle fibers sweeping round behind this to enter the epipericardial ridge. The second shows a little more advance, with distinction of fibers and the line (cl) in which the clavicle is beginning to form: it will later have muscular attachments. The third gives the definitive condition gained by the fixation of muscle to clavicle and the drawing forward of the neck beyond this.

Redrawn from Frazer.[8]

The placodal areas of thickened epithelium appear in association with nerves of the arches. A week later all three borders of the triangular cervical field have encroached upon it, burying the fourth arch but leaving a recess leading to its placodal area (see Fig. 4-B). The placodal areas remain

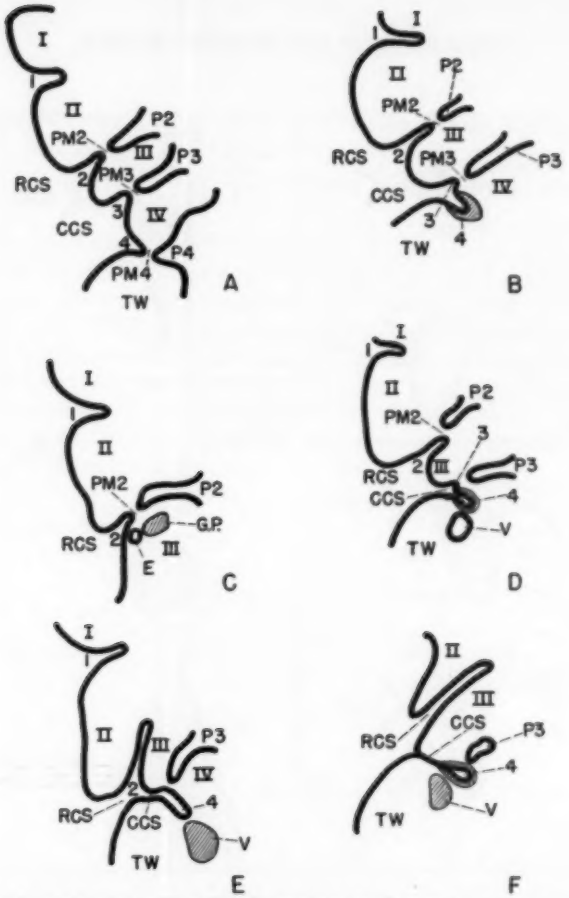

Fig. 7. Redrawn from photomicrographs of Garrett.[8]

A. Human embryo (6 mm.). Section through the pharyngeal region showing the primitive relationships between branchial arches (Roman numerals), clefts (Arabic numerals), and the tips of the corresponding pharyngeal pouches (P 2, 3, 4). x 52.

B. Human embryo (8 mm.). A similar section showing the thickening of the walls of the fourth cleft (4), the regression of the third cleft (3), and the narrowing of the cervical sinus (CCS, RCS). x 52.

C. Human embryo (8 mm.). A section through the same embryo 0.90 mm. nearer the brain, showing an early stage (E) in the formation of vesicle 2. x 52.

D. Human embryo (9 mm.). Section similar to those of Figs. 1 and 2 showing narrowing and deepening of the fourth cleft (4), disappearance of the third cleft (3), and further constriction of the cervical sinus (CCS, RCS). x 52.

E. Human embryo (10.5 mm.). A similar section showing modification of the fourth cleft (4) and constriction of the cervical sinus (CCS, RCS). x 52.

F. Human embryo (12 mm.). Shows the closure of the fourth cleft, transforming it into vesicle 4 (4) by obliteration of the caudal portion of the cervical sinus (CCS). x 52.

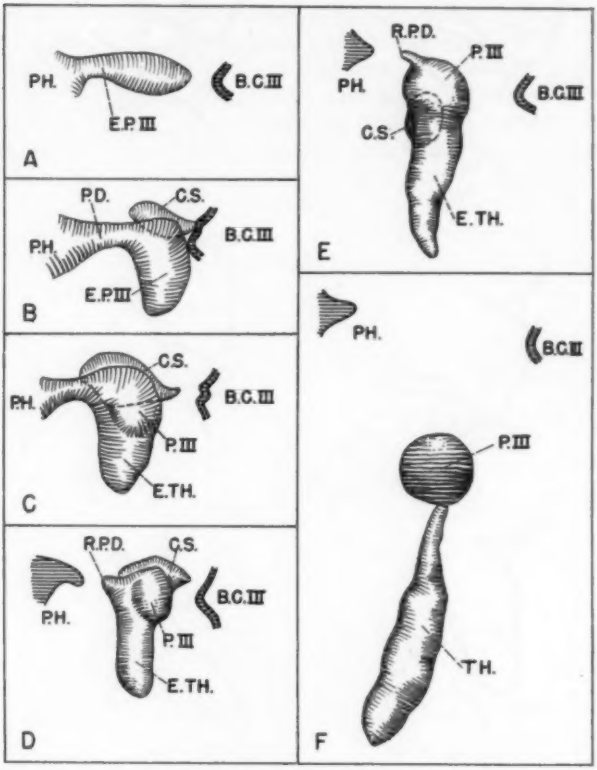

Fig. 8. Schema of the morphogenesis of the third branchial pouch and its derivatives, seen from ventral.

A. Third branchial pouch approaching the floor of branchial cleft III.

B. Elongation of third pouch into the primordium of the thymus. Early association of the cervical sinus with the thymus.

C. Appearance of the primordium of parathyroid III at the cephalic end of the pharyngeal pouch. Separation of ectodermal sinus; its dorsal relation to the upper end of the thymus.

D. Complete separation of third pouch derivatives from the pharynx.

E. Growth of parathyroid III, and elongation of the thymus, beginning of isolation of third branchial complex components.

F. Caudal migration of thymus. B.C., III, Branchial cleft III. C.S., Cervical sinus. E.P., III, Third entodermal pharyngeal pouch. E.Th., Entodermal thymus. P. III, Parathyroid III. P.D., Pharyngo-branchial duct. Ph, Pharynx. R.P.D., Remnant of pharyngo-branchial duct. Th, Thymus.

(Redrawn from illustrations by E. Erickson in article by Norris, 1937. Courtesy, Carnegie Institution of Washington).[49]

connected with the ganglia of their respective nerves, and as growth goes on and the nerves get further from the surface the placodes are left at the bottom of little pits opening on the surface. These disappear very soon.

By the end of the seventh week (12 mm. stage) the fourth arch is covered by the occipital fold. The third arch can be seen on the surface, its groove represented by a faint line (see Fig. 4-C), in the upper part of which is still a connection, the placodal vesicle of the vagus. This arch is clearly marked off in front, for the line of the second outer groove remains as the flexure line of the neck. Thus the "closure" of the cervical sinus is only partial, the third arch remaining flattened down on the surface.

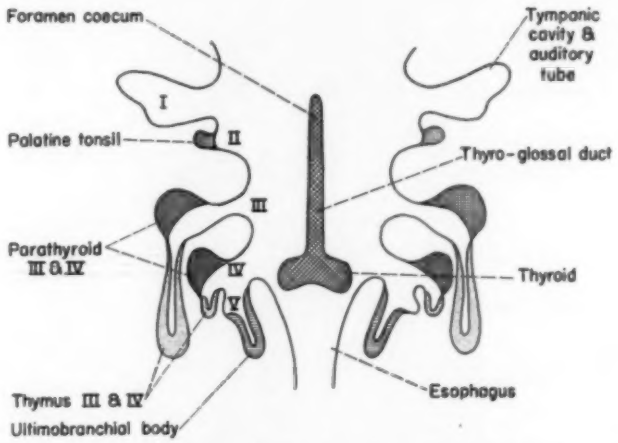

Fig. 9. Diagrammatic outline of the pharynx, at about the stage of 10 mm., to indicate its derivatives. I-V, Pharyngeal pouches.
Redrawn from Arey: "Development Anatomy."[50]

The second arch now becomes appended to the first by virtue of fusion of the walls of the first groove. The upper part of this groove remains as a wide and deep hollow to form the external auditory meatus (see Fig. 4-C) with the wing-like remnant of the second arch forming the lower part of the pinna.

The pharynx in the meantime has been slowly growing away from the pericardium so that the area between the pericardium and the mandibular arch (see Fig. 5) is gradually enlarged with the cells of the epipericardial ridge filling up the interval. Here the muscle cells of this ridge come into contact with the body of the hyoid bone formed between the ventral parts of the second and third arches (hyoid muscles).

The neck comes into evidence as the pharynx separates from the pericardium. The large mandible has the remnant of the second arch (part of the pinna) attached to it, but on a deeper plane are the digastric and stylohyoid muscles, which maintain

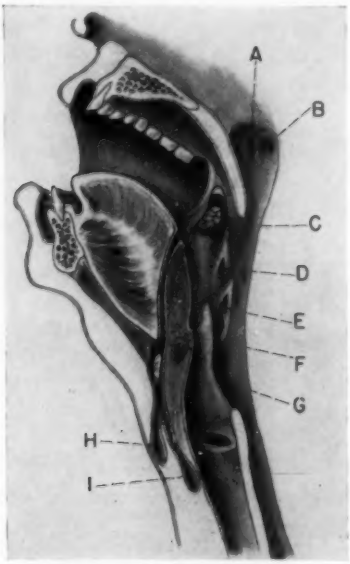

Fig. 10. Diagram showing the position of pharyngeal pouches in the human pharynx. A. Eustachian tube (first pouch); B. Lateral recess of nasopharynx (fossa of Rosenmueller); C. Tonsil (second pouch); D. Position of third pouch, according to Keith (73); E. Position of third pouch according to Arey (2); F. Elevation over internal laryngeal nerve; G. Position of fourth pouch according to Arey (2); H. Median thyroid forming cyst and fistula; I. Median thyroid forming isthmus.

the original position of this arch. Behind them is the flexure line of the neck, and then the area which was originally the external third arch which now enlarges to extend over the

anterior triangle of the neck. This is bounded ventrally by infrahyoid muscles, and behind by the sternomastoid, derivatives respectively of the epipericardial structures and the caudal limiting ridge (see Fig. 6).

In an earlier report Frazer depicts the cervical sinus drawing away from the pharynx with continued growth, leaving long cell-strands of ectoderm in contact with the pharyngeal pouches (see Figs. 14, 15). The cervical sinus is connected to

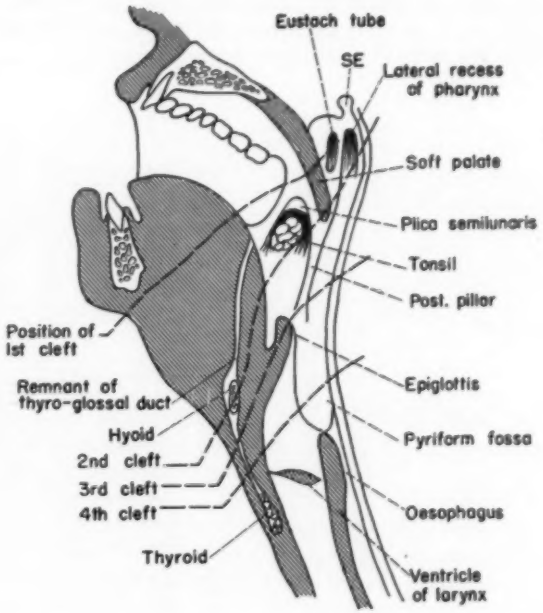

Fig. 11. Showing the position of the Visceral Clefts in the adult. The lines only indicate the approximate positions of the clefts. For instance, the soft palate is made up largely from the third arch. SE = median pharyngeal recess or bursa.

Redrawn from Keith: "Human Embryology and Morphology."[7]

the external surface by a narrow tract. The external duct running from the region of the second pouch opens into this tract and not directly into the cervical sinus.

ENTODERMAL ASPECT AND THE PHARYNGEAL POUCHES.

The pharyngeal pouches have disappeared from the interior of the pharynx by the end of the second month, with the exception of the upper part of the first, which forms the Eustachian tube, tympanic cavity and mastoid air-cell system. The external auditory meatus is formed from the first groove. The tympanic membrane remains in the position of a cleft or closing membrane. The lateral pouches have dorsal and ventral angles, and the surface of contact with the ectoderm extends from one angle to the other[1] (see Fig. 14). In the case of the first pouch there is no definite ventral angle, and the epithelial contact is only with the upturned dorsal angle.

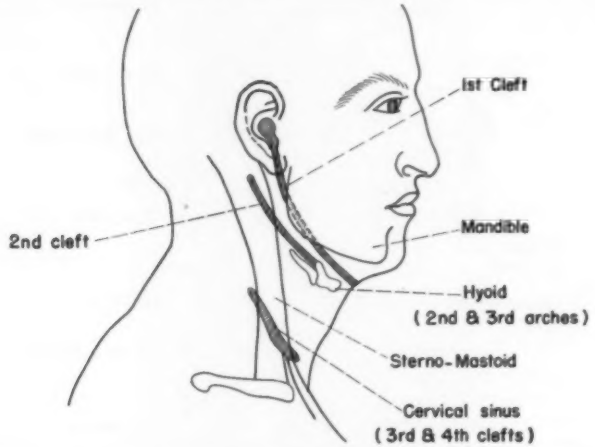

Fig. 12. Showing the position of the External Cleft Depressions in the Adult.
Redrawn from Keith: "Human Embryology and Morphology."[7]

The second pharyngeal pouch is obliterated ventrally by the development of the tongue and by a proliferation of its endodermal lining. Only a small portion may persist as a small recess (tonsillar fossa). The dorsal portion of this pouch may contribute to the corresponding tubo-tympanic recess.[2] The distance between the angles in the depth of the second pouch increases rapidly, the upper angle and the pouch below it lose contact with the ectoderm and come to lie permanently in

the middle ear[1] (see Fig. 14). The lower or ventral angle of the second pouch is located in the tonsillar fossa.

The pyriform fossa, anterior to the fold of the internal laryngeal nerve, marks the site of the third pharyngeal pouch in the adult. The fourth pharyngeal pouch opening site lies behind the fold over the internal laryngeal nerve[4] (see Fig. 10).

PATHOLOGICAL CONSIDERATIONS.

The pathogenesis of lateral cervical cysts and fistulas still remains widely disputed chiefly because of the various opinions concerning the embryonic development of the neck.

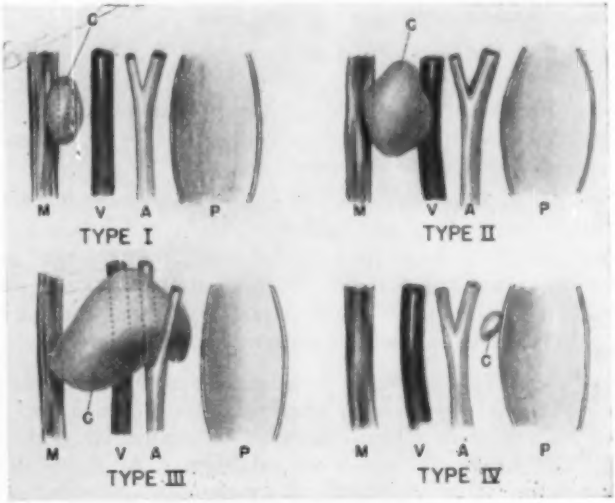

Fig. . Diagrams showing the relationship of Branchial Cysts to the surrounding structures. They are of four types.
Redrawn from Bailey: "Branchial Cysts."[6]

Most authorities adhere to the branchiogenic theory on the basis of one of the following:[5]

1. Vestigial remains (epithelial rests) of the branchial apparatus.

2. Incomplete closure of the branchial grooves.

3. Rupture of the closing membrane separating the branchial grooves from the branchial pouches.

4. Anomalous development of the branchial apparatus.

5. Persistence of a cervical sinus.

It is usually assumed that fistulas are the result of incomplete closure of a branchial groove or pouch, or both, and that cysts are a result of vestigial epithelial remnants (see Figs. 11, 12, 16, 17, 18).

Bailey[6] has described four types of branchial cysts based on their relations to surrounding structures (see Fig. 13):

Type I — This is the type which lies superficially immediately beneath the cervical fascia and at the anterior border of the sternomastoid muscle.

Type II — This is the commonest variety. It is beneath the enveloping fascia. It extends down to and lies on the great vessels. The wall must be carefully separated from the internal jugular vein to which it is varyingly adherent. Fascial pressure causes them to become oblong.

Type III — Extension inward to the lateral wall of the pharynx occurs. The cyst passes between the internal and external carotids. In addition a prolongation of the cyst may pass upward as far as the lateral mass of the atlas and even to the base of the skull. The overlying portion of the sternomastoid muscle is thinned and flattened out over the cyst.

Type IV — This is usually a columnar-lined cyst lying adjacent to the pharyngeal wall, and medial to the carotids. As a rule this type is seldom observed clinically, since it lies deeply and usually produces no symptoms. At times these cysts develop after tonsillectomy as the post-operative scar closes a previously patent and unnoticed internal incomplete branchial fistula.

Frazer[8] was of the opinion that Bailey's *Type I* originated from the external tract leading to the cervical sinus or from the second external duct which runs into this tract (see Fig. 14). *Type II* is ascribed to an enlargement of a persistent cervical sinus. *Type III* is referred to as an enlargement of the second external duct perhaps combined with an enlarged cer-

vical sinus. *Type IV* may be the remnant of an internal pharyngeal duct, or may possibly be derived from one of the "epithelial bodies" which are found in association with the pouches; or, in spite of its epithelium it may be derived from ectoderm.

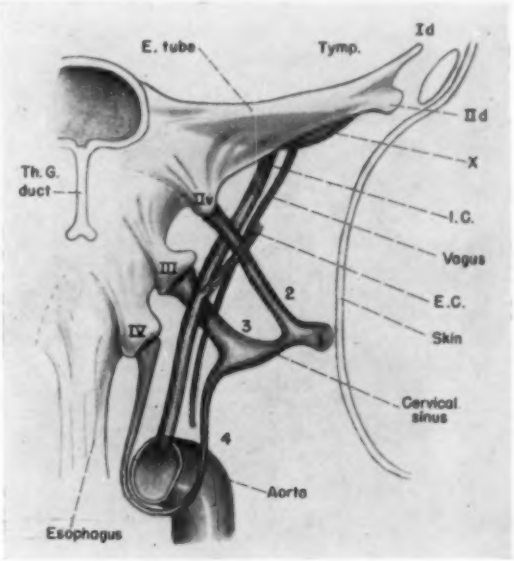

Fig. 14. Schematic figure to show the vestigial structures in the neck, with their relations to main arteries and nerves. The epithelial bodies are not represented. Id IId, dorsal angles of 1st and 2nd lateral pouches. IIv, III, IV, "internal pharyngeal ducts" derived from ventral angles of 2nd, 3rd and 4th lateral pouches. 2, 3, 4, "external pharyngeal ducts" derived from 2nd, 3rd, and 4th external grooves. X. Layer of entodermal cells cut off from the lower part of Eustachian tube. E. Tube Eustachian tube. E.C. External carotid. I.C. Internal carotid. Th.G. Duct. Thyroglossal duct.

Redrawn from Frazer.[1]

According to Frazer,[1] there is only one way of settling the origin of any particular instance of persistent and enlarged vestige, and that is by a careful anatomical examination with reference to relations, followed after removal by microscopic investigation. The anatomical relations are most important in their pharyngeal aspects. "The three pouches and their corresponding external ducts bear definite and distinct rela-

tions to the main vascular and nervous structures, and these relations, if the vessels are normal, are absolutely fixed and certain" (see Fig. 14).

It will be observed that the second external duct passes between the two carotids and in front of the vagus. The third

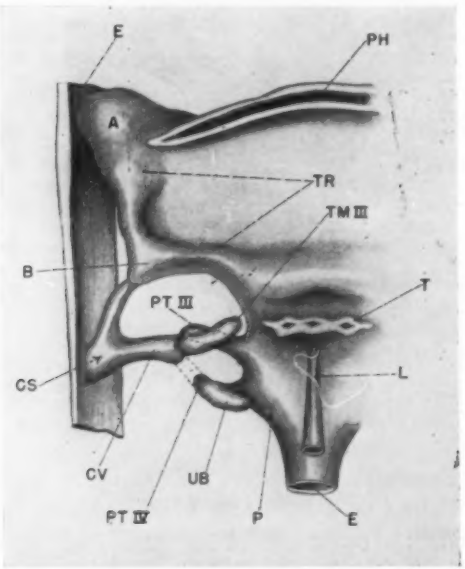

Fig. 15. The Lining Membrane of the Pharynx of a Human Embryo at the end of the sixth week of development (10 mm.) viewed on its ventral aspect (Grosser). E. Ectoderm. A. First pocket. B. Second pocket (tonsil). CS. Cervical Sinus. CV. Cervical vesicle. PT IV. Parathyroid IV. UB. Ultimobranchial body (thyroid). E. Esophagus. L. Larynx. P. Pyriform fossa. T. Thyroid. TM III. Thymus III. PT III. Parathyroid III. TR. Tubotympanic recess. PH. Pharynx.
Redrawn from Keith: "Human Embryology and Morphology."[7]

external duct goes behind the common or internal carotid and in front of the vagus and reaches the interior of the pharynx below the hyoid. The fourth external duct is drawn down by the arch of the aorta on the left side and by the subclavian on the right. If the closing membrane were perforated these ducts might lead into the pharynx; the second into the tonsil,

the third into the pyriform fossa anterior to the fold of the internal laryngeal nerve, and the fourth into the pyriform fossa behind the fold of the internal laryngeal[4] (see Figs. 10, 11, 12).

The cervical sinus lies superficial to the great vessels, anterior to the sternomastoid and below the posterior belly of the digastric. Fistulas of the second groove, or cleft, and

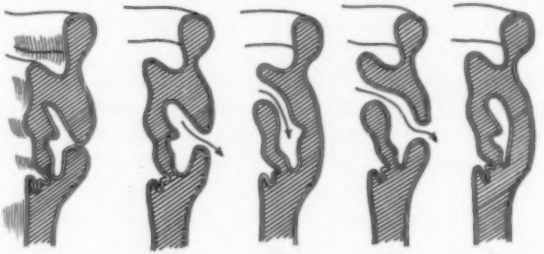

Fig. 16. Series of diagrams to show how several kinds of tracts and cysts may arise through faulty development of the pharyngeal wall.

A. Normal pharynx showing closure of cervical sinus.

B. Incomplete closure of cervical sinus forming the basis for a tract opening externally upon the surface of the neck.

C. Rupture of closing membrane leaving permanent opening into the position of the second pouch.

D. Branchial fistula resulting from a combination of the conditions in B and C.

E. Cystic remnant of the cervical sinus.

Redrawn from Ward and Hendrick. "Tumors of the Head and Neck."[15]

pouch have been recognized on many occasions. The ectodermal derivatives might be expected to have a stratified cell lining and the entodermal prolongations a columnar celled epithelium. Frazer states that lymphadenoid tissue of the walls of cysts may presumably be thymic.[1] In some lower mammals a "superficial thymus" is found in association with the ectoderm of the cervical sinus.

An interesting observation of Frazer's is the presence of a double layer of entodermal cells cut off from the lower aspect of the Eustachian tube (see Fig. 14). "It is the result of the

same process which has separated the dorsal and ventral angles of the second pouch. A forward growth from the third arch destroys this portion of the second pouch and comes up against the first groove, which is caught between it and the first arch." This double layer of entodermal cells disappears quickly. The possibility of its persisting as a vestigial remnant is suggested by Frazer. It would lie below the Eustachian tube, behind the tensor palati and in front of the carotid and stylopharyngeus. "If by any chance it opened into the pharynx, it would do so through the sinus of Morgagni."[1]

Keith indicates that if there is a residual of placodal or of any developmental pocket it should appear along the anterior border of the sternomastoid. He further states that the cutaneous opening often contains a tag of skin representing a rudimentary external ear containing a piece of cartilage. The dorsal part of the second cleft may persist, an auricle becoming developed on it, while the normal external ear is suppressed.[7]

The sources for vestigial remnants in the neck are the placodal cysts and their ducts, persistence of one or more of the ectodermal "external pharyngeal ducts" which are connected with the entodermal pouches, and abnormal fusion between the walls of the second groove in some parts of its extent. Every vestigial remnant in the lateral neck that has a deep connection as well as an outer opening passes upward to cross the hypoglossal nerve before running to its original deep attachment.[8]

Fistulas of branchiogenic origin are divided into three types (see Fig. 16):

1. Incomplete external fistula — has an external opening but no internal communication with the pharynx.

2. Incomplete internal fistula — opening into the pharynx but not externally.

3. Complete fistula — complete tract with both internal and external openings.

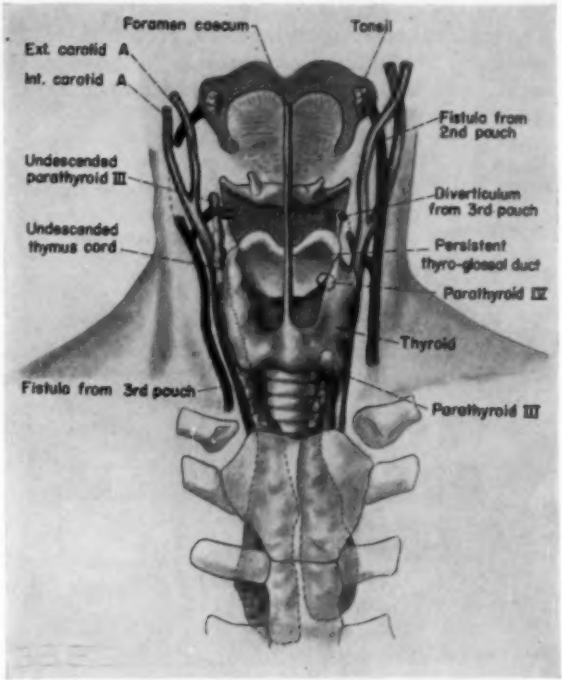

Fig. 17. Schematic diagram in which is collected a number of anomalous conditions resulting from the faulty development of the cervical sinus, pharynx, and glandular derivatives of the pharyngeal epithelium. On the subject's right: note undescended parathyroid III, residual thymus cord, and a branchial fistula from pharyngeal pouch III. A previous connection of the fistula with a diverticulum of the supratonsillar fossa is broken. Center: persistent thyroglossal duct passing ventral to (or through) the hyoid bone. On the subject's left: note a diverticulum of pouch III, an incompletely descended thymus still attached to parathyroid III, and a fistula of second pouch origin.

Redrawn from Braus: "Anatomie des Menschen," Bd. 2.

The embryonic thymic duct may give rise to persistent anomalies.[9] A fistula of the second groove can be distinguished from a fistula of the thymopharyngeal duct on the following basis:

1. A fistula of the second groove must pass between the internal and external carotids. Most complete fistulas do this. A fistula of the thymopharyngeal duct does not pass between the carotids but lies anteriorly to and very close to the carotid

sheath. Very few, if any, complete branchial fistulas do this.

2. The internal opening of a second groove fistula is in the tonsil fossa.[10] The internal opening of a thymopharyngeal duct fistula is into the region of the pyriform fossa which very rarely occurs.

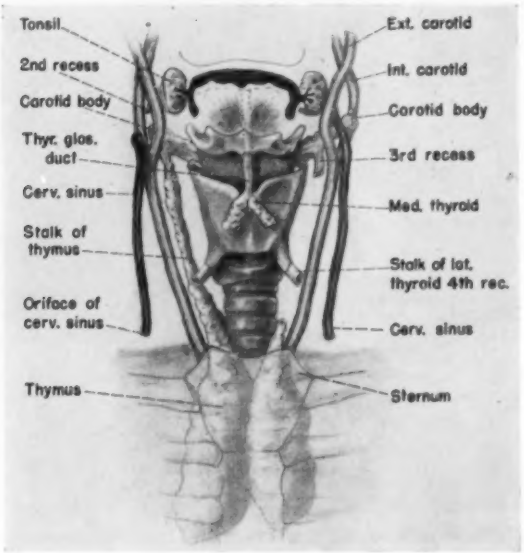

Fig. 18. Diagram to illustrate the various parts of the visceral recesses and grooves which may persist. The second recess gives rise to the tonsil; the recess may be in contact with an epithelial tube derived from the cervical sinus. The third recess gives rise to the thymus and parathyroid III. The carotid body arises at the site of the third recess, but its development has nothing to do with the recess.

Redrawn from Keith: "Human Embryology and Morphology."[7]

3. The location of the external opening is not of value in differentiating the two types, since either can open along the entire anterior border of the sternomastoid muscle.

4. If a cyst wall contains typical thymic tissue with Hassall's corpuscles it may be of thymopharyngeal duct origin. These are rare. There is a possibility of thymic remnants in the neck becoming associated with a second groove fistula. If

a cyst or fistula does not contain thymic tissue in its walls it is probably of branchiogenic origin.

5. Both types contain lymphoid tissue elements.

6. Most cysts and fistulas contain squamous epithelium. Some contain columnar or ciliated epithelium. There is no reason why a thymic tract should contain a squamous epithelium.

7. If the remnants of a thymic tract are responsible, cystic structures should occur anywhere from the final resting place of the thymus to the site of its origin.

8. The relationship of complete branchial fistulas to the stylopharyngeus muscle is rather vaguely reported. The stylopharyngeus is a derivative of the third arch. It is directed downward much more than forward; it is a thin, narrow and long muscle which requires meticulous dissection to distinguish it from the other muscles in the region. Theoretically a second groove and pouch fistula should pass anterosuperiorly to this muscle on its way to the supratonsillar fossa.

9. Branchial structures may occur above the hyoid bone and above the jaw. It would be difficult to explain the constant occurrence of lymphoid tissue, presumed by Wenglowski to come from the thymus, in the walls of fistulas and cysts in these locations.[11]

10. It is difficult to explain the inconstant position of the external openings of fistulas and the relatively high locations of most branchial cysts with Wenglowski's theory.[11]

LATERAL CERVICAL OR BRANCHIAL CYST.

History and Symptoms:

The most common symptom is a painless swelling in the neck (see Fig. 19), usually below the angle of the jaw and along the anterior border of the sternomastoid muscle. The swelling may become quite large before it is noticed. This is probably because the swelling is gradual as a rule. Occasionally the swelling is rapid, particularly after an injury to the neck. Not infrequently the patient states that the swelling varies in size from day to day. This is most likely due to absorption

of the cystic fluid through the walls of the cyst. In a few cases there may be a communication into the pharynx through which the cystic fluid may escape. Some patients state that the cyst

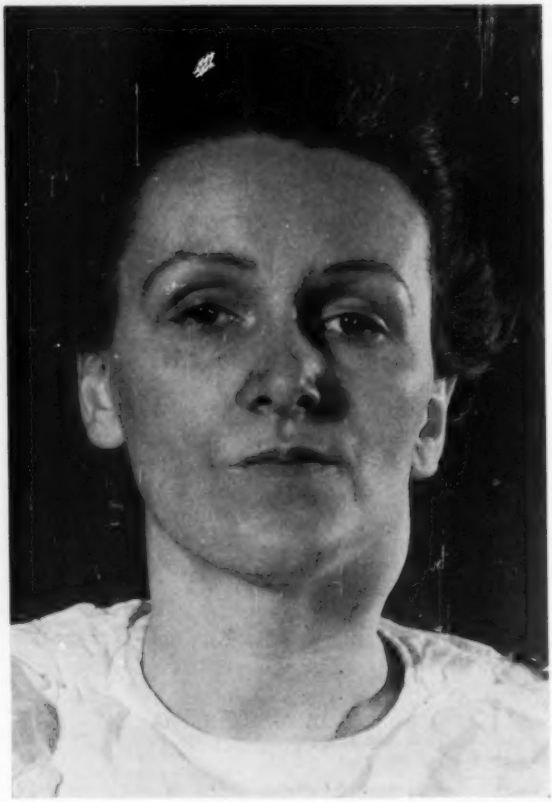

Fig. 19. Typical appearance of the usual branchial cleft cyst.

becomes tense at times, and it is in this group that some pain may be noted.

Very large cysts may give rise to a sensation of pressure, fullness or drawing. These may have dysphagia, huskiness

and bulging into the floor of the mouth. They rarely bulge into the pharynx.

Males and females are almost equally affected. The ages vary from extreme youth to old age. There is a familial tendency to branchial cysts.

PHYSICAL SIGNS.

The size of the cyst varies from that of an olive (1.5x2.5 cm.) to the size of a large orange. (10 cm.) The actual size of a cyst cannot always be ascertained clinically.

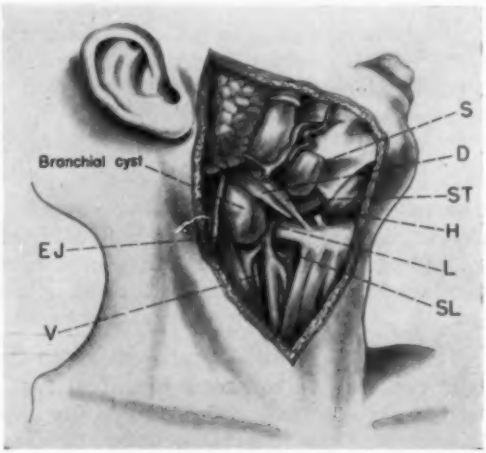

Fig. 20. Drawing of a usual branchial cyst with its normal relations. S. Submaxillary gland. D. Digastric muscle. ST. Stylohyoid muscle. H. Hypoglossal nerve. L. Lingual artery. SL. Superior laryngeal nerve. V. Vagus nerve. E.J. External jugular vein.

The swelling is cystic, and fluctuation is usually elicited. In large tense cysts which have thick walls, cheesy contents, and are deep seated, this sign may be absent. In these cases this sign may again be sought for by having the swelling "fixed" by an assistant and manipulating in two planes at right angles to each other.

Cysts are usually not translucent. Occasionally a cyst originating from a pharyngeal pouch, lined by columnar epi-

thelium, filled with mucus, and having reached a large size may become translucent. Cysts are movable, not fixed to underlying structures and skin, regularly outlined, and covered with smooth skin unless there has been considerable inflammatory reaction around the cyst.

Cysts lie in relationship with the deep surface of the upper half of the sternomastoid (see Fig. 20). It nearly always protrudes around the anterior border of this muscle and rarely around its posterior border. Its center usually is opposite the great horn of the hyoid. The sternomastoid is frequently thin and flattened out over a cyst, so that it is difficult to palpate. One can overcome this by standing behind the patient and asking him to push his chin as hard as possible against your palm. This tightens the sternomastoid so that it can be palpated from low in the neck (where it is normal) upwards along its anterior border.

DIFFERENTIAL DIAGNOSIS.

With a complete examination and with an adequate working knowledge of the various pathological lesions in the neck the clinician can diagnose a large percentage of branchial cysts. Frequently, however, the final diagnosis must be made by the pathologist.

Tuberculous Lymph Glands — Tuberculosis in the cervical lymph nodes frequently simulates a branchial cyst. The contents of a cold abscess frequently look like the contents of a cyst. Bailey[6,12,13] claims that tubercle bacilli are found in only 10 per cent of tuberculous glands. The contents of both cavities, then, are usually sterile. All groups of cervical lymph nodes should be palpated from behind the patient whose head is bent forward. The complete absence of cervical adenitis is in favor of the swelling being a branchial cyst. It is unlikely, but not impossible, that a tuberculous abscess would exist for many months without involving the skin.

Cystic Hygroma — A cystic hygroma is translucent while a branchial cyst is opaque. The former, usually located in the lower half of the neck, is diffuse, loculated and first noted in infancy. A branchial cyst is well circumscribed, unilocular and is usually located in the upper part of the neck.

Solitary Lymph Cyst — These are invariably translucent and located in the supraclavicular triangle. These are probably related to cystic hygroma but usually appear in adult life.

Hemangioma and Lymphangioma — The cavernous hemangioma is bluish in color and presents a "bag of worms" sensation on palpation, due to the enlarged tortuous veins. Bailey[6] suggests using the "sign of emptying." If the cystic swelling is a venous hemangioma then pressure will cause it to decrease in size and when pressure is removed it slowly refills. It is possible for a large branchial cyst with deep ramifications to give this sign by emptying into the retropharyngeal space; hence, when exerting this pressure, the pharynx should be watched before pronouncing the "sign of emptying" positive.

Cystic Degeneration of a Malignant Neoplasm — A rapidly growing primary or metastatic malignancy in the "branchial position" may become cystic.

Carotid Body Tumor — Branchial cysts are much more common. These tumors occur at the carotid bifurcation. They move laterally but not vertically. Pulsation is usually transmitted. It is important to distinguish between these two lesions, because removal of a carotid body tumor is associated with tremendous risks.

Dermoid Cyst — These contain hair. They are superficial and are never attached to the lateral pharyngeal wall.

Primary Lymphomas — This may lead to confusion, especially if there is only one enlarged node. The lymphomas are rarely single and often appear as a group of fused, adherent nodes. Lymphomas are apt to be firmer than branchial cysts.

Aberrant Thyroid — These may occur at any level from the hyoid to the suprasternal notch. They vary in size from 1 to 5 cm. In general they are firm, mobile and are not cystic.

Neurofibroma — These are occasionally seen in the neck. They may present a rather firm tumor that is immobile and varies from 1 to 4 cm. in diameter. They may or may not be multiple.

Aneurysm — Aneurysm of the carotids usually bulge into the pharynx. They do not necessarily transmit a pulsation since the pulse pressure is low within. Aspiration with a fine needle would confirm the diagnosis. If in doubt carotid arteriography would be diagnostic.

Lipomas — These fatty tumors are frequently lobulated and may suggest a branchial cyst. Aspiration of course would not yield fluid.

Suppurative Cervical Lymphadenitis — Acute inflammation of a branchial cyst may simulate a suppurating cervical lymph node. The history of a previous swelling in the neck would speak for a branchial cyst.

Chronic Retropharyngeal Abscess — Especially in cases of tuberculosis. A branchial cyst may present in the pharynx, extend up to the atlas and outward to the great vessels.[14]

Thyroglossal Duct Cyst — These need not be in the midline. They are often on the left side but not so far laterally as the sternomastoid. They are attached to the hyoid and will move up and down with swallowing.

Myxomatous Degeneration of a Mixed Parotid Tumor — An intraparotid branchial cyst was described by Fredet[15] and by Cunningham.[16] These evidently arose from the first cleft.

Accessory Thymus — These are rare remnants found along the course of the thymopharyngeal duct. They may be associated with aberrant parathyroid tissue.[17]

PATHOLOGY.

The contents of a cyst lined by squamous epithelium is an opaque fluid which passes readily through an aspirating needle. If this fluid is placed in a dish and moved to and fro the shimmer of its lipoid contents will be noticed.[6] If the fluid is dropped on a slide and covered with a cover-slip and examined under low power, numerous cholesterol crystals will be seen, which is characteristic of these cysts. At times the contents of a cyst consists of a cheesy material suggesting tapioca granules.[18]

Those cysts lined with a columnar epithelium have a thick, sticky, mucoid fluid which is transparent.

Microscopic Pathology — Microscopically branchial cysts usually have a stratified squamous epithelium, which is surrounded by lymphoid tissue and a layer of loose connective tissue. In 263 of the 287 specimens reported by Neel and Pemberton the lining was a squamous cell epithelium. In 18 cases it was columnar and in six cases both types of epithelium were represented. The columnar epithelium may contain cilia (see Fig. 22). The supporting layer of connective tissue in these cysts occasionally contains serous glands and muscle tissue. Evidence of chronic inflammation is almost always present in the walls of these cysts. Accidental or intentional incision of a cyst is likely to result in an acquired branchial fistula. Occasionally a cyst is reported to contain thymic tissue in its wall. Such a cyst was reported by Rudberg.[19]

BRANCHIOGENIC CARCINOMA.

Many authorities are of the opinion that branchiogenetic carcinoma does not exist. This diagnosis can be considered only after a thorough search of the mouth, nasopharynx, tonsil, larynx, hypopharynx and external auditory canal. Crile and Kearns[20] stated that less than 100 cases had been reported up to 1935. In the large series of branchial cysts (287 cases) reported by Neel and Pemberton[21] they found no suggestion of carcinoma in any specimen.

TREATMENT.

The only treatment that will effect a cure is complete surgical excision. In the event of acute suppuration surgery must be delayed until the inflammatory reaction has subsided. If the suppuration does not immediately respond to conservative measures, incision and drainage might be necessary leaving the ultimate excision to a later date.

The use of sclerosing solutions, repeated aspirations and X-ray therapy are mentioned only to be strongly condemned. They seldom produce a cure, and they cause unpredictable inflammatory reactions, which make subsequent surgical extirpation much more difficult.

The cyst is exposed through a single transverse incision following the line of the creases in the neck. The incision

should be near the middle of the cyst and the flaps of skin and platysma dissected from the underlying enveloping fascia. If the cyst is unusually large the sternomastoid muscle may be divided to afford proper exposure.

The underlying spinal accessory nerve should be carefully isolated. It bears a posterior relationship to the cyst wall. The branchial cyst may run deeply in the neck and extend

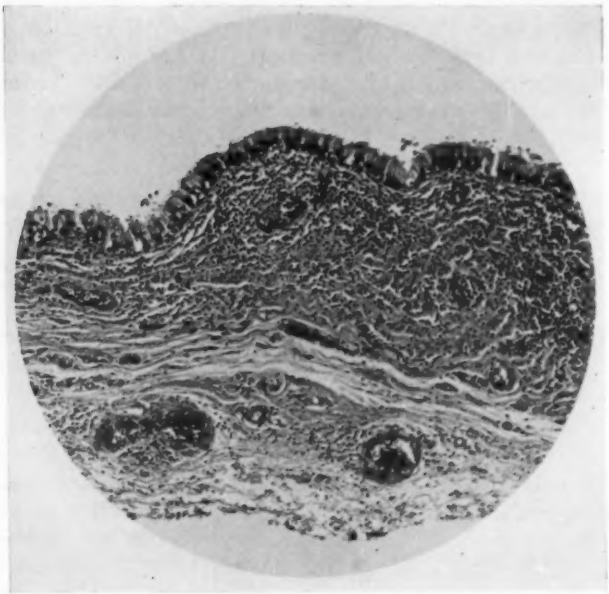

Fig. 22. Photomicrograph of a branchial cyst which was found behind the posterior tonsil pillar. Note the ciliated columnar epithelium.

upward as far as the base of the skull. It is highly desirable to remove the cyst intact. If it bursts half way through the operation dissection becomes difficult, surrounding structures become endangered, and the end result may be unsatisfactory, for even a small piece of secreting epithelium left behind may give rise to a sinus which persistently discharges.

The following technique simplifies the operation and makes intact removal almost always possible. The superficial aspect of the cyst is cleaned and about half of the liquid contents of the cyst aspirated with a small needle and saved for reinjection into the cyst after removal so as to check for the possibility of overlooking a fistulous tract. The puncture hole is then covered with a small piece of gauze which is then seized along with the underlying collapsed cyst wall, with a sponge forceps.[6] The gauze helps to keep the forceps from slipping. Gentle traction is made with the forceps in various directions, while the cyst wall is dissected out by blunt dissection with gauze, forceps or scissors. Great care must be taken not to overlook a pedicle which must be removed along with the cyst.[22] The pedicle may extend to the tonsil fossa and may be so narrow that it is difficult to dissect. Extensive dissection may be required.

LATERAL CERVICAL FISTULAS.

The typical cervical fistula is present at birth and has its external opening a short distance above the sternoclavicular joint and in front of the sternomastoid muscle. The fistulous tract is usually palpable as a firm cord which passes upward under the platysma muscle and enveloping fascia and superficial to the sternohyoid and sternothyroid muscles (see Fig. 23). The course parallels the sternomastoid muscle. It is in close relation with the underlying carotid artery and internal jugular vein. At the level of the hyoid bone it turns inward, crosses over the hypoglossal nerve, passes beneath the posterior belly of the digastric muscle, passes between the internal and external carotid arteries and over the glossopharyngeal nerve. It then passes beneath the styloglossus muscle and above the stylopharyngeous muscle. It finally extends upward along the posterior aspect of the tonsil into the posterior palatine arch to end in the upper half of the posterior faucial pillar, the supratonsillar fossa,[23,24,25,26,27] or it may extend through the tonsil opening directly on its surface.[19,21,28,29]

According to Rudberg[19] about 90 per cent of fistulas show squamous epithelium, 8 per cent ciliated columnar, and 2 per cent show both types. When the lining is columnar there is often a persistent, annoying secretion of mucus.[6] When the

fistula is of a uniformly large caliber ingested liquids may leak through. If the fistula is of thymogenic origin then thymic tissue will be found along it,[9,23] on microscopic examination.

Many times the external orifice is small and inconspicuous.

In the series of 239 cases of lateral cervical cysts and fistulas reported by Neel and Pemberton[21] 80 were fistulas. Of these 40 were external fistulas, 31 complete fistulas and nine internal fistulas. Fifty-eight complained of a draining sinus. The

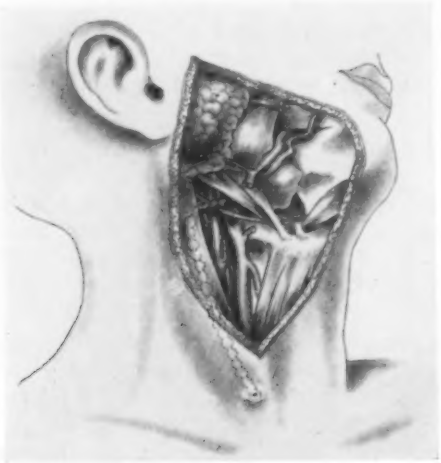

Fig. 23. Drawing of a complete fistula of second groove and pouch origin with its usual relations. Note the fistula passing upward under the sternomastoid muscle. At the level of the hypoglossal nerve it bends almost at right angles over the nerve and between the internal and external carotids to reach the region of the pharyngopalatine arch a short distance further. Not shown is the passage of the tract over the glossopharyngeal nerve and stylopharyngeus muscle which lie deep to the carotids.

majority of external fistulas were produced by ill-advised incision of a cyst (acquired fistulas) which was mistaken for an abscess. Semken[30] stated that complete fistulas are present in approximately one-third of reported cases. When a complete fistula is present the external orifice can be seen to rise with deglutition.

The internal opening of a complete or of an internal fistula can be found in only a small number of these cases. If an internal fistula is associated with a cyst, the cyst may fill with food, air or liquids when swallowing, or it may become infected and fill with pus. Pressure applied to such a cyst may express its contents into the pharynx. This situation is sometimes initiated by tonsillectomy.[11,21]

Fistulas may be unilateral or bilateral. They may occur in several members of the same family.[11,21,32] Fistulas occasionally have accessory tracts or diverticulums[11] which must not be overlooked in surgery. When a fistula is probed such symptoms as pallor, palpitation, coughing, and vomiting may occur, due to the close proximity of the tract to the vagus nerve.[32,33]

A fistula of the third pharyngeal pouch should open into the pharynx in the plane of the thyrohyoid membrane. These are extremely rare.

An internal fistula may open into the trachea or larynx and give rise to air sacs and present the features of what has been called "aerial goitre," "aerial bronchocele," "tracheocele," and "hernia of the trachea."[18,32] These should be differentiated from a laryngocele.

A tuberculous fistula must be considered in the differential diagnosis. The congenital fistula is present at birth whereas the tuberculous one is not. The course and extent of a fistula can be demonstrated by the injection of the tract with a radio-opaque oil and taking a Roentgenogram (see Fig. 24). A congenital fistula will reveal a smooth wall whereas a tuberculous fistula will be very irregular.[34] If the shadow extends to the pharynx a diagnosis of a complete fistula can be made.

TREATMENT.

Total surgical extirpation offers the only sure cure. Previous inflammatory reactions may make the dissection of a tract difficult. Orientation as to the extent of the tract may be facilitated by the injection of the tract with methylene blue and then washing it out with saline.[35] Enough dye remains on the wall to stain it but not enough to stain the operative field. A blunt needle and a minimal of pressure is required for this

injection. The tract may be injected with liquid paraffin, which solidifies and makes the dissection easier.[36] A catheter can be inserted into the fistula as a guide for dissection.[37] McNeil Love[38] advocates a procedure whereby the orifice of the fistula is encircled with a fine purse-string suture which is

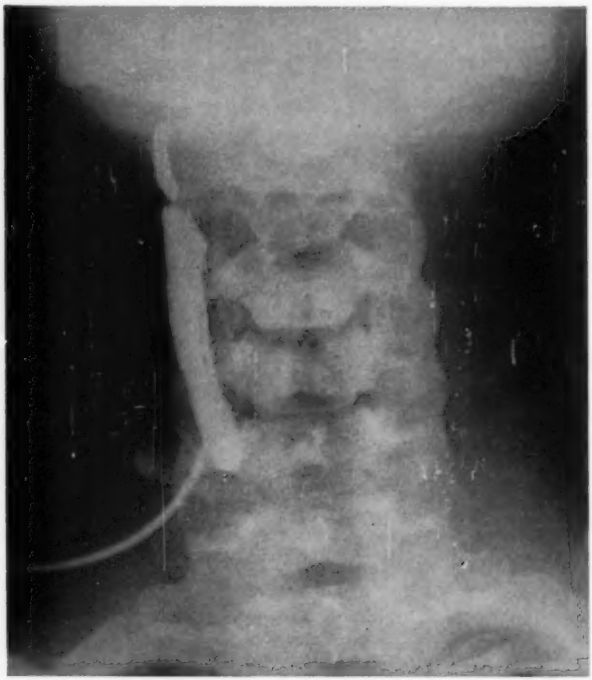

Fig. 24. Case 29. Complete fistula injected with lipiodol. Note upper fourth of the tract is narrow and communicates via a much narrower and shorter tract with a large dilated inferior portion. Swinging laterally is a catheter.

inserted as close to the orifice of the fistula as possible and then tightened to close the orifice. In a few days continued secretion distends the fistula, transforming it into a cyst, which thus facilitates dissection. The extent of the tract is determined by the injection of a liquid radio-opaque material

(see Fig. 24)[39] such as lipoidol under local anesthesia (infiltrated around the external opening). A purse-string suture

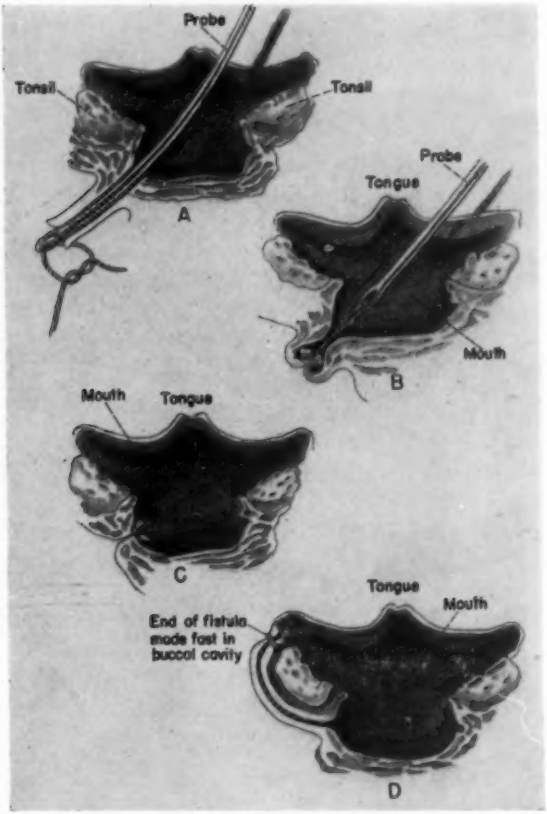

Fig. 25. Cross Sections at Tonsil Level. Diagram to show the steps in the von Hacker and Koenig operations. A. The dissected fistulous tract has been amputated and the stump has been made fast to the eye of the probe. B. Probe drawn into mouth and fistula being inverted. C. Tract has been entirely inverted, ligated and cut off. D. Procedure in the Koenig operation. The stump of the tract has been brought anterior to the tonsil and passed into the buccal cavity where it has been made fast.

Redrawn from Christopher.[51]

is placed around the fistula before a blunt needle or catheter is inserted into the tract. As soon as the injection is completed

the syringe and its attachment are withdrawn and the purse-string suture tied to prevent the escape of the radio-opaque material before the X-ray pictures are made.

Sclerosing solutions have been advocated by some au-

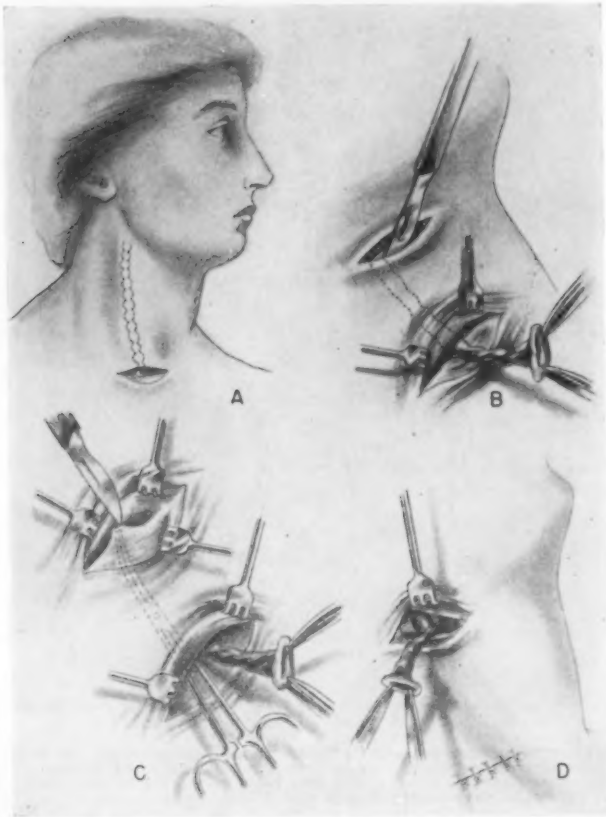

Fig. 26. The step-ladder operation—A. Transverse incision about the external orifice of the fistula. B. Tract partially freed and the second incision made at the hyoid level. C. Fistulous tract dissected from below up to the second incision. The envesting fascia is cut. D. The fistulous tract is brought out through the second incision. From this level the dissection is carried down to the termination of the fistula in the supraton-sillar fossa. Note the fistula passing over the hypoglossal nerve and inward between the carotids.

Redrawn with modifications from Baumgartner.[52]

thors[40,41,42,43]. Beck[41] used this method in treating fistulas in children. It has an advantage because a scar is avoided. The step-ladder technique, to be described later, leaves very little scarring, however. With the injection treatment there is the danger of marked inflammatory reaction and necrosis with perforation into the pharynx and hence is not recommended.

Von Hacker[44] described the following method of surgical removal (see Fig. 25 A-C). The orifice of the tract is circumscribed and the tract dissected upward through an incision along the anterior border of the sternomastoid. Above the bifurcation of the carotid the tract is carefully followed between the carotids and below the digastric to the lateral wall of the pharynx through which it passes to the tonsillar region. As it may be difficult to follow the course of the fistula between the carotids the tract may be opened at this site and a probe carefully inserted to locate the opening in the pharynx. The probe is then advanced until it can be seized through the mouth. The fistulous tract is then fastened about the probe by means of a silk suture at a point central from the site of the perforation. The probe is then slowly and carefully withdrawn through the mouth, the fistulous end fixed to the probe is slowly invaginated, and the fistula is loosened from its attachments. The end of the fistula may in this way be brought through the pharynx without difficulty. The rest of the tract is ligated with catgut near its base and resected above this ligature.

If the fistula cannot be dissected to the pharynx, perhaps as a result of previous inflammation, Koenig's technique[45] (see Fig. 25-D) may be used whereby the distal end of the fistula is brought into the buccal cavity in front of the tonsil so that the secretions drain into the mouth instead of externally.

Koenig's operation is as follows: The tract is dissected as high up toward the digastric as possible and then by blunt finger dissection another passageway to the pharynx is created. A probe is inserted through the neck wound into the depth of the new passage until it engages the mucosa as the base of the anterior tonsil pillar. The mucosa at this point is incised and the probe is pushed through into the pharynx. The wall of the dissected branchial fistula is fastened to the fenestrated end of the probe with a suture and is drawn

through the opening into the lumen of the pharynx. It is thus led through, but not turned inside out, as in Von Hacker's operation. The fistula is pulled tight and sutured to the mucosa. The fistula projecting into the pharynx is cut off, leaving a short stump which curves around the base of the tonsil. The original distal end of the fistula is in the supratonsillar fossa, and the amputated anterior end of the fistula

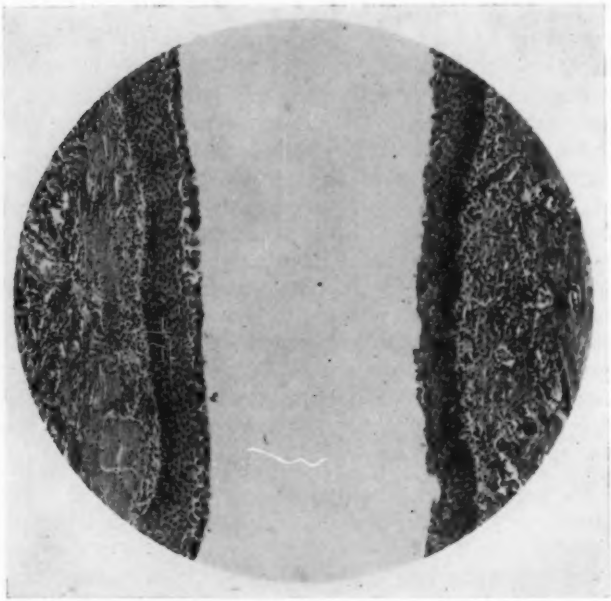

Fig. 27. Photomicrograph of branchial fistula lined with stratified squamous epithelium.

is in the newly made opening at the base of the anterior tonsil pillar. Koenig's and Von Hacker's operations are recommended if the fistula is large. If a stump is ligated and left in the wound a recurrence may take place in the form of an internal fistula which can discharge into the pharynx.

The most satisfactory operation for removal is the step-

ladder technique[46,47] (see Fig. 26). An elliptical transverse incision is made about the external orifice of the fistula. Gentle traction is applied to the fistula while dissection is carried up beneath the skin, platysma muscle and fascia. A second transverse incision is made over the fistulous tract at a higher

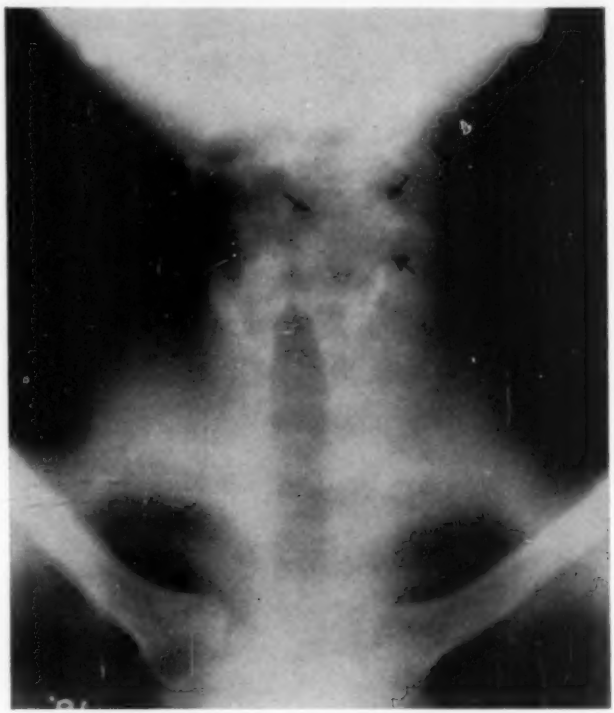

Fig. 28. Laminogram of larynx showing position of branchial cyst of third branchial pouch origin (arrows).

level. The tract is then threaded through from the first to the second incision and the dissection resumed from this second level until the tract is completely removed. When the tract approaches the pharynx an assistant places a finger in Rosenmueller's fossa and presses down and laterally to facilitate fol-

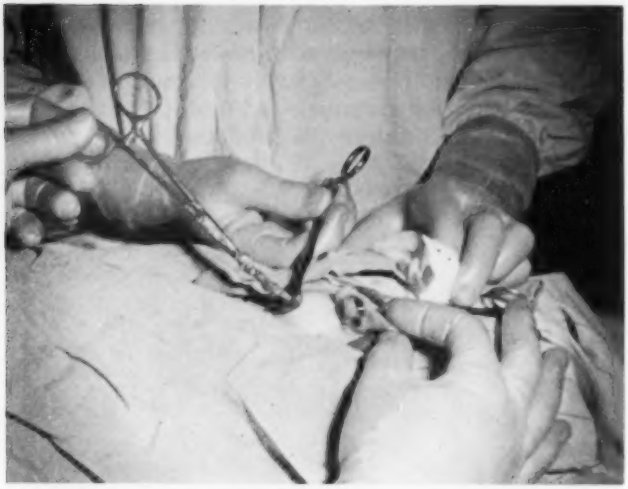

Fig. 29. Showing the step-ladder technique in the case of a placodal fistula of the second arch. Note the vestigial elevation on the posterior surface of the left auricle which is partly circumscribed by incision. The transverse and eliptical incision lower in the neck is opposite the greater horn of the hyoid and includes abnormal skin around the lower orifice of the fistula. The tract has been dissected upward where it was found to pass under the facial nerve to which it was firmly adherent. The tract was exposed again through another incision at a point intermediate between two incisions seen in the photograph. After freeing the fistulous tract from under the facial nerve it was dissected upward and completely removed along with the fistula and tumescence on the posterior surface of the auricle.

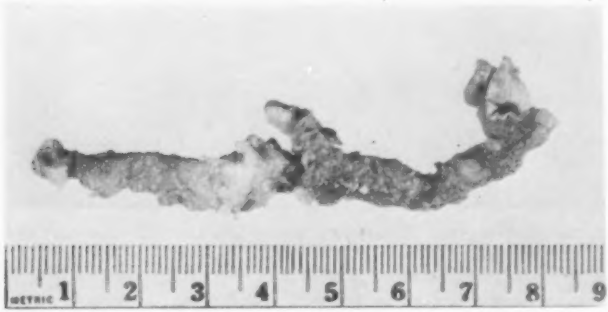

Fig. 30. Photograph of fistulous tract, the position which was shown in Fig. 29. At the first cm. level is the lower external orifice opposite the greater horn of the hyoid. Between the 3½-5 cm. level is seen irregularly attached cartilage. At the 6½ cm. level is seen the groove created by passage of the fistula around the facial nerve. At the 7 cm. level an annular ring of cartilage is seen which was adherent to the inferior aspect of the auricular cartilage. At the 7½-8 cm. level one can observe the external orifice of the upper end of the tract.

lowing the tract into the pharynx.[18] The tract is ligated with fine silk or catgut at its entrance into the pharynx. The external incisions are closed with fine interrupted silk sutures. A long drain is placed through the lower incision and left in place several days. With this technique the scars are much less noticeable than when a long incision is made along the anterior border of the sternomastoid.

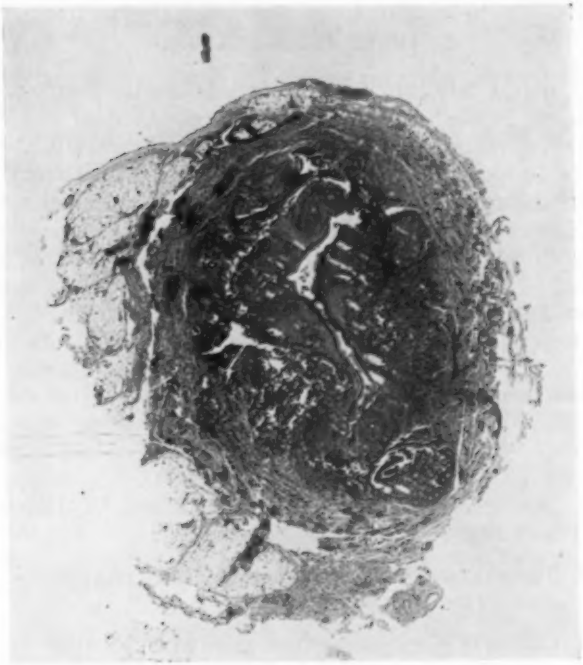

Fig. 31. Photomicrograph of fistulous tract in Fig. 29. Note lumen lined by stratified squamous epithelium. Note the layer of lymphoid tissue and the external collagenous supporting structure.

When removing a complete fistula tonsillectomy can be performed simultaneously,[23,28] before[24] or afterward.[19,25]

An intimate knowledge of the embryology and anatomy of the neck is required for the surgical removal of cervical

anomalies.[19] These anomalous structures are closely related to vital structures. Their dissection at times is tedious and intricate and requires patience and skill. In the event of a second operation the skill required is even greater. It must also be remembered that these anomalies need not always be extirpated: if the cyst or fistula is symptomless there is no reason for operation, and the risk of later malignant degeneration is probably of no importance.[39]

SUMMARY AND CONCLUSIONS.

1. Lateral cervical cysts and fistulas are the result of disturbances in the early embryonic development of the neck. These disturbances are due to the failure of complete absorption of included ectodermal and entodermal epithelium that is buried during the morphogenesis of the branchial arches.

2. The various embryonic sites where anomalies may develop are somewhat as follows:

a. The visceral or branchial pouches;

b. The branchial clefts or grooves;

c. The cervical sinus;

d. The epithelial placodes which are associated with the ganglia of the nerves of the arches;

e. Persistence of cervical vesicles II and IV (Garrett) (see Fig. 7);

f. The pharyngobranchial duct III (third pharyngeal duct) (see Fig. 8);

g. Canals of Kursteiner which arise when parathyroid III separates from the cervical thymus (see Figs. 8, 9).

3. If pharyngeal pouches persist their internal openings would be as follows:

a. Second pouch (dorsal or upper angle) orifice in lateral wall of nasopharynx (above the palato-pharyngeal arch).

b. Second pouch (lower or ventral angle) orifice in the

region of the supratonsillar fossa. This is the most common type of pouch anomaly.

c. Third pouch orifice in the lateral pharyngeal wall or pyriform fossa above or anterior to the internal laryngeal nerve. It may penetrate the thyrohyoid membrane.

d. Fourth pouch orifice in lateral pharyngeal wall or pyriform fossa below or behind the internal laryngeal nerve.

4. Cysts in the lateral pharyngeal wall may be the remnant of an internal pharyngeal duct, or may be derived from one of the epithelial bodies which are found in association with the pouches.

5. Abnormal fusion between the walls of branchial clefts or grooves may give rise to branchial cysts and fistulas. This is particularly common in the second cleft, which is probably the source of most of the usual branchial cysts. Those cysts found in intimate relation with the tail of the parotid, rather than with the carotid sheath, are probably of first cleft origin. The external openings of branchial grooves, when they persist, are very variable in their position due to rapid growth of the ventral neck during the period in embryonic life when the branchial arches are developing. When present they are along the anterior border of the sternomastoid muscle, particularly in the case of second cleft or cervical sinus anomaly. Congenital auricular fistulas, usually attributed to first cleft anomaly, actually arise from the developing skin of a specific intertubercular groove.[48]

6. Failure of obliteration of the cervical sinus through fusion of its walls may be responsible for some branchial cysts; however, it must be kept in mind that in normal development no part of the cervical sinus, as separate from grooves, forms a closed cyst-like structure.

7. The epithelial placodes which are associated with the ganglia of the nerves of the arches may persist as placodal cysts which draw away from the pharynx with continued growth, leaving long cell-strands of ectoderm in contact with the pharyngeal pouches.

8. The pharyngobranchial duct III (third pharyngeal duct) may fragment irregularly, leaving small isolated fragments which may later differentiate into accessory parathyroid or thymic rudiments, or may even be the site of origin of epithelial cysts.

9. When parathyroid III separates from the cervical thymus small epithelial tubules arise which may persist (canals of Kursteiner) and give rise to cysts associated with parathyroid III and thymus III.

10. The cervical sinus epithelium does not contribute to the formation of the thymus, which originates from the entoderm of the third pharyngeal duct.

11. The embryonic thymic duct may give rise to persistent anomalies, but these are rare.

12. The origin of any particular vestige can be deducted only after a careful anatomical examination with emphasis on relations followed after removal by microscopic examination. These anatomical relations are most important in their pharyngeal aspects. The second, third and fourth pouches and their corresponding external ducts bear definite and distinct relations to the main vascular and nervous structures.

13. Every vestigial remnant in the lateral neck that has a deep connection as well as an outer opening passes up to cross the hypoglossal nerve before running to its original deep attachment.

14. The symptomatology, physical findings, differential diagnosis, pathology and treatment of branchial cysts and fistulas is reviewed.

15. The surgical treatment of lateral cervical anomalies requires an intimate knowledge of the embryology and anatomy of the neck if one is to avoid damage to important structures and avoid the risks of recurrence.

BIBLIOGRAPHY.

1. Frazer, J. Ernest: The Nomenclature of Diseased States Caused by Certain Vestigial Structures in the Neck. *Brit. Jour. Surg.*, 11:131-136, 1923.

2. Frazer, J. Ernest: A Manual of Embryology, 2nd Ed. Bailliere, Tindall and Cox, London, 1940.

3. Garrett, F. D.: Development of the Cervical Vesicles in Man. *Anat. Record*, 100:101-113, 1948.

4. Krahl, V. E.: The Early Development of the Head and Neck, Chapter II in Ward and Hendrick: Tumors of the Head and Neck. Williams and Wilkins, Baltimore, 1950.

5. Malcolm, R. E., and Benson, R. E.: Branchial Cysts. *Surg.*, 7: 187-203, 1940.

6. Bailey, H.: Branchial Cysts. H. K. Lewis and Co., London.

7. Keith, Sir Arthur: Human Embryology and Morphology, 6th Ed. Edward Arnold and Company, London, 1948.

8. Frazer, J. Ernest: The Disappearance of the Precervical Sinus. *Jour. Anat.*, 61:132-143, 1926.

9. Wengelowski, R.: Ueber die Halsfisteln un Cysten. *Arch. F. Klin. Chir.*, 98:151, 1912 and 10:789, 1913.

10. Kostanecki and Mielecki, A.: Die Angeborenen Kiemenfisteln de Menschen. *Arch. f. Path. Anat. u. Phys.*, 120:385-436 and 121:55-87, 1890.

11. Carp, L., and Stout, A. P.: Branchial Anomalies and Neoplasms: A Report of 32 Cases with Follow-up Results. *Ann. Surg.*, 87:186-209, 1928.

12. Bailey, H.: The Clinical Aspects of Branchial Cysts. *Brit. Jour. Surg.*, 10:565-572, 1923.

13. Bailey, H.: Clinical Aspects of Branchial Fistula. *Brit. Jour. Surg.*, 21:173-182, 1933.

14. Coakley, C. G.: A Branchial Cleft Cyst Simulating a Chronic Retropharyngeal Abscess. *Trans. Amer. Laryngol. Assn.*, 26: 19-27, 1904.

15. Fredet, P., and Chevassu, M.: Epithelioma Branchial Intraparotidien. *Bul. Med. Soc. Anat.*, Paris, 77:621-633, 1902.

16. Cunningham, W. F.: Branchial Cysts of the Parotid Gland. *Ann. Surg.*, 90:114-117, 1929.

17. Lange-Hellman, J. E.: Accessory Thymus Tissue of the Neck. *Acta Otolaryngol.*, 42:375-378, 1952.

18. Ward, G. E., and Hendrick, J. W.: Tumors of the Head and Neck. Williams and Wilkins, Baltimore, 1950.

19. Rudberg, R. D.: Congenital Fistulae of the Neck. *Acta. Otolaryngologica*, Supplement 116, 271-283, 1954.

20. Child, G., and Kearns, J. E., Jr.: Branchial Carcinoma: Lateral Cervical Neoplasm *Surg., Gynec. Obstet.*, 60:703-709, 1935.

21. Neel, H. B., and Pemberton, J.: Lateral Cervical (Branchial) Cysts and Fistulas. *Surg.*, 18:267-286, 1945.

22. Brown, J. Mackenzie: Branchial and Thyroglossal Duct Cysts and Fistulas. *Ann. Otol. Rhinol. and Laryngol.*, 44:644-653, 1935.

23. Gill, N. W.: A Branchial Fistula Terminating in a Tubulodermoid Cyst Lying Posterior to the Tonsil. *Jour. Laryngol and Otol.*, 64:62-69, 1950.

24. Hall, C.: Surgical Removal of Congenital Neck Fistula. The Laryngoscope, 60:368-375, 1950.

25. Rehak, P.: Beitrag zur Operation der Lateralen (Branchiogenen) Halszyste. *Monatsschr. fur Ohrenh.*, 84:137, 1950.

26. Gillman, P. K.: Branchial Cysts and Fistulas. *Jour. A.M.A.*, 77: 26-28, 1921.

27. Taylor, J. N. S., and Burwell, R. G.: Branchogenic Nasopharyngeal Cysts. *Jour. Laryngol. and Otol.*, 68:667-679, 1954.

28. Blassingame, C. D.: Congenital Cysts and Fistulae About the Head and Neck. *Ann. Otol. Rhinol. and Laryngol.*, 56:395-403, 1947.

29. Kleinert, M. H.: Branchial Fistula. *Arch. Otolaryngol.*, 18:510,515, 1933.

30. Semken, G. H.: Branchiogenetic (Lateral) Fistulae and Cysts, in Nelson New Loose-Leaf Surgery, Vol. 2. Thos. Nelson and Sons, New York, pp. 783-795, 1937.

31. Kramer, R.: Lateral Cervical Fistulae. The Laryngoscope, 36: 517-522, 1926.

32. Hyndman, O. R., and Light, G.: The Branchial Apparatus. *Arch. Surg.*, 19:510-452, 1929.

33. Carp, L.: Branchial Fistula; Its Clinical Relation to Irritation of the Vagus. *Surg. Gynec. Obstet.*, 52:772-777, 1926.

34. Wagensteen, O. H.: Differentiation of Branchial from other Cervical Cysts by X-ray Examination. *Ann. Surg.*, 93:790-791, 1931.

35. Baumgartner, C. J.: Congenital Lesions of the Neck. *Jour. A.M.A.*, 113:1860-1864, 1939.

36. Cristopher, F.: Lateral Cervical Fistula; Paraffin Injection as An Aid to Excision. *S. Clin. North America*, 10:351-353, 1930.

37. Dowd, C. N.: Complete Branchiogenic Fistula. *Ann. Surg.*, 63:519-522, 1916.

38. Love, R. J. McNeil: A Simple Method of Dealing with Congenital Fistula. *Lancet*, 217:122, 1929.

39. Andreassen, M.: Congenital Cysts and Fistulas of the Neck. *Acta. Otolaryngoligca*, Suppl., 109:15-19, 1953.

40. Cutler, E. C., and Zollinger, R.: The Use of Sclerosing Solutions in the Treatment of Cysts and Fistulae. *Amer. Jour. Surg.*, 19:411-418, 1933.

41. Beck, W. C.: Branchiogenic Cysts in Infancy. *Surg.*, 1:792-799, 1937.

42. Lendgren, C. V.: Branchiogenic Sinus: Treatment by Injection. *Ann. West. Med. and Surg.*, 2:463-464, 1948.

43. Berry, G.: Branchial Cyst: Two Instructive Cases. *Ann. Otol. Rhinol. and Laryngol.*, 43:287-294, 1934.

44. Von Hacker: Extirpation der Kompleten Seitlichen Halsfisteln Mittels Extraktion des Oberen Strangendes von der Mundhohle aus. *Zentrabl. f. Chir.*, 24:1073-1076, 1897.

45. Koenig, F.: *Arch. f. Klin. Chir.*, 51:578, 1895.

46. Broca, A.: Quoted by Christopher.[36]

47. Douglas, J.: Branchiogenic Cyst with Sinus Leading into Pharynx. *Ann. Surg.*, 67:240-241, 1918.

48. Congdon, E. D.; Rowhanavegse, S., and Varamisara, P.: Human Congenital Auricular and Juxta; Auricular Fossae, Sinuses and Scars Including the So-called Aural and Auricular Fistulae) and the Bearing of their Anatomy upon the Theories of their Genesis. *Amer. Jour. Anat.*, 51:439-463, 1932.

49. Norris, E. H.: The Parathyroid Glands and the Lateral Thyroid in

Man: their Morphogenesis, Histogenesis, Topographic Anatomy and Prenatal Growth. *Carnegie Cont. to Embr.*, 26:247-294, 1937.

50. Arey, L. A.: Developmental Anatomy, 2nd Ed. W. B. Saunders Company, Philadelphia, 1930.

51. Christopher, F.: The Surgical Treatment of Lateral Cervical Fistulae. *Surg., Gynec. Obstet.*, 38:329-335, 1924.

52. Baumgartner, C. J.: Lateral Cysts and Fistulas of the Neck. *Calif. Med.*, 73:466-472, 1950.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The Sixth International Congress of Otolaryngology will take place in Washington, D. C., from Sunday, May 5, through Friday, May 10, 1957, under the presidency of Arthur W. Proetz, M.D. The subscription for Members is $25.00 (U.S.A.) which will include all official meetings of the Congress except the banquet. Ladies and other relatives accompanying members may be registered as Associates at a fee of $10.00.

The selected subjects for the Plenary (Combined) Sessions to be held Monday, Wednesday and Friday mornings will be:

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2. Collagen Disorders of the Respiratory Tract.
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Two types of communications are invited: *1.* Contributions to the discussions of the selected subjects, limited to five minutes. *2.* Original papers, limited to 15 minutes. These should be in one of the four official languages: English, French, German, Spanish.

Motion picture films will be shown continuously except during the Plenary Sessions. There will be both scientific and technical exhibits.

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PRESBYCUSIS.*

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Detroit, Mich.

In the cochlea, as in all the other organs of the body, there are senescent changes with the passage of time. The classical manifestation of cochlear aging is high tone deafness caused by atrophic changes in the basal coil.

The purpose of this report is to present the histological findings in the cochlea of several animals and one human, all of whom had such high tone hearing losses. The findings are particularly significant because there is a minimum of superimposed postmortem autolytic change.

THE AGING PHENOMENON.

The high tone deafness of aging is the cochlear manifestation of a diffuse process which involves all the body tissues. Within the fourth decade of life there already is decreased efficiency of some organs and of the individual as a whole. In the fifth decade there usually is an apparent reduction in efficiency, and the further passage of time brings a cascade of senile changes which terminate in death during the seventh or eighth decades of life. Many investigations have been performed in an attempt to provide information on the mechanism of the aging process. It is known that with increasing age there is decreased ability of certain cell systems to undergo mitosis.[1,2] decrease in nucleoprotein in certain systems,[3] accumulation of insoluble compounds in the cytoplasm,[4] and chemical changes occur in the intercellular fluid.[5] A great many morphological, chemical and physiological changes have been demonstrated in the individual cells of the various body systems.[6]

It is now clearly understood that aging cannot be accounted for on the basis of changes in one particular system, *e.g.*, the

*Read as Candidate's Thesis, at the Fifty-ninth Annual Meeting of the American Laryngological, Rhinological, and Otological Society, Inc., Hollywood, Fla., March, 1955.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, February 10, 1955.

cardiovascular system in man. Although popular cliché has it that a man is as old as his arteries, there is not a satisfactory parallelism between vascular change and senility. Too many symptoms of old age are apparently independent of blood vessel alteration to establish this single entity as a cause of senility. The study of aging changes in individual cells is complicated by the fact that the cellular population changes during the life of the body and also that there is difficulty in distinguishing the normal changes of aging from alteration caused by injury or disease.

Cowdry[5] has helped to clarify this complex problem by classifying the cells of the body into four groups, depending upon the character of their lives: *Group 1*, termed "vegetative intermitotics," includes those cells whose main function is reproductive, such as the red bone marrow, the basal cells of the epidermis and the spermatogonia of the testicles. *Group 2*, "differentiating intermitotics," consists of a group exhibiting increasing specialization, such as the lineage of cells which lead to leukocytes and spermatozoa. *Group 3* is designated as "reverting postmitotics" because, though they generally age and die, some of them are capable of reversion. Liver cells, for example, can multiply in the event of the removal or death of large numbers of their fellows. *Group 4* is called the "fixed postmitotics" and constitutes the highest plane of differentiation. After specialized function has been established for them there is no turning back; they age and die. Among this group are cardiac and skeletal muscle cells, odontoblasts, bone cells, rod and cone cells, cells of the organ of Corti, nerve cells including the spiral ganglion, and dozens of other cell types. The deafness of aging is caused by degenerative changes occurring in fixed postmitotic cells. For them the length of individual cell life is not conditioned by stimulation or inhibition of mitotic activity but by ability to maintain their characteristic structural organization in the continual adaptation they must make to changes of the tissue fluid environment. The length of individual life for many postmitotic cell types seems to be determined in some measure by heredity. It is known that certain families display hereditary patterns of premature cellular decay in certain

cell systems; *e.g.*, high tone deafness is known to develop earlier in some families than in others.

EXISTING CONCEPTS REGARDING PRESBYCUSIS.

Since Zwaardemaker in 1899[7] first described the clinical manifestations of high tone deafness with aging there have been a number of reports on the pathology of this condition. Some of the changes described undoubtedly were due to post-mortem autolysis of the membranous labyrinth.

An outstanding contribution to our knowledge of the pathology of high tone deafness was made by Crowe, Guild and Polvogt in 1934.[8] This study was a correlation of the clinical and pathological findings in 79 human ears, the audiograms of which showed impairment of hearing limited to the high tones. Although there were many exceptions, they were able to classify their cases into two general groups: one with abrupt high tone hearing losses in which the most prominent lesion was an area of atrophy of the organ of Corti at the basal turn; and a second with gradual high tone loss in which the most prominent lesion was partial atrophy of the cochlear nerve supply in the basal turn. In some cases they were unable to explain the hearing losses on the basis of cochlear pathology. They found no parallelism between arteriosclerotic changes in the vessels of the ear and degeneration of the membranous labyrinth and nerve, and discounted arteriosclerosis as a cause of the changes.

Saxen[9] presented another important paper in 1937 on the pathological changes in human cochleae as the result of aging. He found two types of degenerative change: *1.* Atrophy of the spiral ganglion, beginning in the basal coil, and regarded by him as process of wear and tear typical of old age; *2.* Angiosclerotic degeneration of the inner ear, characterized by degenerative change of the epithelial tissues of the membranous cochlear labyrinth and considered by him to be due to arteriosclerosis. He also had an elderly patient with marked hearing loss and no labyrinthine or cochlear nerve changes and believed, therefore, that deafness could result from changes in the central nervous system.

Guild and Saxen both recognized two types of presbycusis:

the pathology in one involving mainly the organ of Corti; in the other, the spiral ganglion. They also believed that there were some cases in which there was inadequate cochlear pathology to explain the deafness. The relationship of these two types of presbycusis to each other and to aging processes elsewhere in the body has not been clarified.

METHOD.

In a number of experiments during the past few years my associates and I have examined the cochleae of 72 cats, on which we performed tests of auditory threshold. Behavioral tests were performed on 27, cortical tests on 32, and both cortical and behavioral tests on 13. Three of these 72 animals (4 per cent) had objectively demonstrable hearing losses prior to experimentation and on histological examination were found to have degenerative lesions in the basal coil. For the remaining 69 ears the thresholds were normal for the frequency range 125 to 16,000 cps. If we subtract from this group the 22 ears with experimental pathological lesions in the basal coil and 21 ears from very young animals there remain 26 cochleae from full-grown cats without an experimental lesion in the basal coil and with normal thresholds up to 16,000 cps. Five, or 20 per cent of these cochleae had degenerative changes in the basal coil ranging from one-half mm. to two and one-half mm. in size. These lesions, obviously, were too far basalward to register as a hearing loss on our tests.

An additional ear exhibiting a high tone threshold loss on cortical testing was provided by Dr. Joseph Hind.

For all animals the function of one ear was destroyed by a surgical operation. The behavioral hearing tests were performed by training animals to respond to pure tone auditory stimuli by moving forward in a rotating cage to avoid shock. When the response was learned the stimulus intensity was reduced to determine auditory thresholds. The cortical test was performed by exposing the contralateral auditory cortex and displaying the potential difference between an electrode on the cortex and an electrode on the neck on a cathode ray oscilloscope. A determination was made of the minimum sound pressure required at each frequency to elicit strychnine

spikes to a tone pulse delivered to the ear through a piece of plastic tubing. Both the behavioral and cortical methods are described in detail elsewhere.[10,11]

The animals were sacrificed by intravital perfusion with 10 per cent formalin solution. Postmortem autolysis was arrested in the human ear by injecting 20 per cent formalin into the middle ear soon after death. The temporal bones were decalcified, imbedded in celloidin and serially sectioned. Graphic reconstructions of the cochleae were made and the pathological findings entered on tables. Quantitative estimates of degree of pathological change were made for 11 structures at approximately 125 points in each cochlea.

The threshold curves and the pathological findings in the cochleae have been graphically displayed on charts. The frequency scale of each audiogram and the distance scale of the cochlear chart were plotted on parallel and equal length coordinates. The frequencies were arranged according to the anatomical frequency scale which takes into consideration their spatial distribution along the cochlear duct. The evidence supporting this scale for the cat is presented elsewhere.[10] The black filling in the cochlear charts indicates the estimated percentage of degenerative change for the various structures.

PRESBYCUSIS DUE TO EPITHELIAL ATROPHY.

The pathological findings in four cat ears are representative of the first type of presbycusis which is characterized by atrophic changes in the membranous labyrinth (see Figs. 1 and 2).

Cat 1. This animal was sacrificed by intravital perfusion with 10 per cent formalin after having been used for another, unrelated, experiment. The behavioral thresholds were within the range of average normal for the frequencies tested from 125 to 16,000 cps. The pathological findings in the cochlea are representative of those changes occurring in a series of five animals having no demonstrable threshold changes but having small lesions at the basal end of the cochlea.

There was atrophy of the organ of Corti in the first 2.5 mm. with total loss of hair cells and partial loss of spiral ganglion cells in this region. Lesser degrees of atrophy existed in the pillars to the 3.8 mm. region and in Hensen's and sulcus cells to 4.5 mm. The tectorial membrane was markedly distorted in the first 2.5 mm. The stria vascularis, limbus, spiral ligament and Reissner's membrane were normal throughout. There were no detectable changes in the vessels of the stria vascularis, spiral ligament or modiolus. The efferent nerve bundle appeared normal. No attempt was made in this study to evaluate the condition

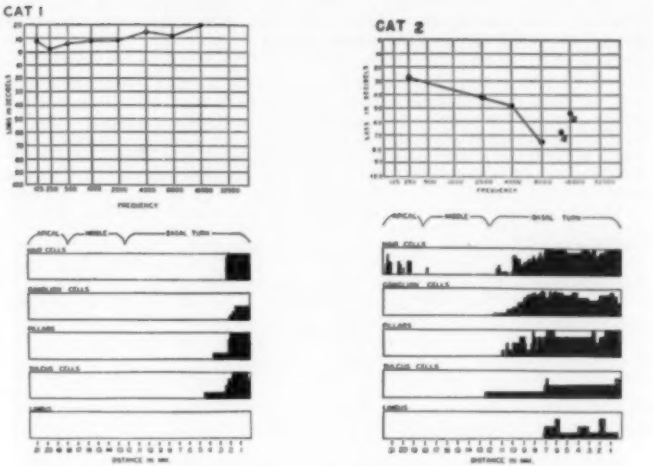

Fig. 1. Behavioral audiograms and cochlear charts for two cats with epithelial atrophy of basal end of cochlea.

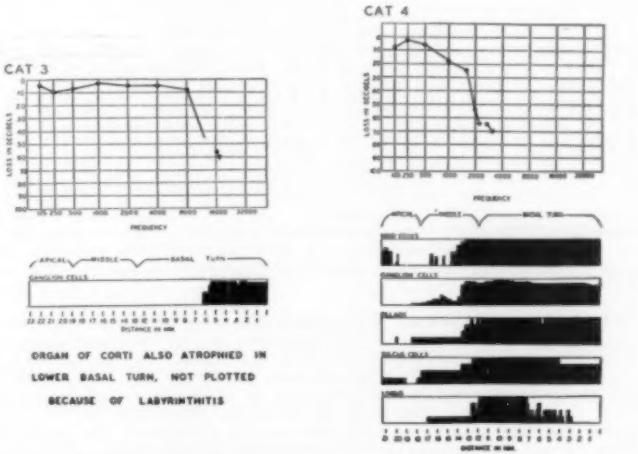

Fig. 2. Behavioral audiograms and cochlear charts for two cats with epithelial atrophy of basal end of cochlea.

of the efferent nerve fibres in the basal 4 mm. of any of the cochleae studied. The vestibular membranous labyrinth, including the sense organs, and vestibular nerves were normal.

Cat. 2. The behavioral audiogram on this animal revealed a gradual type of high tone threshold loss. The animal was given a general anesthetic and an otoscopic examination was performed to rule out the possibility of impacted cerumen as a cause for the hearing loss. The animal was sacrificed by intravital perfusion with 10 per cent formalin. The middle ear, auditory bulla and ear drum appeared normal. Histological examination showed a close parallelism in the severity of atrophic change occurring in hair cells, ganglion cells and pillar cells. The change in sulcus cells and Hensen's cells extended 1 mm. further than other detectable changes in the membranous labyrinth. Decreased cellularity was noted in the limbus from .5 mm. to 7 mm., and in the spiral ligament from 0 to 8 mm. There was localized atrophy of the stria in the 2 to 4 mm. region. The tectorial membrane and Reissner's membrane appeared normal throughout. There were no detectable changes in the blood vessels of the spiral ligament, stria vascularis, or modiolus. The efferent nerve bundles to the basal turn (Bundle "B" and "C" of Rasmussen[12]) were severely degenerated, whereas the efferent bundle to the middle and apical turns (Bundle "A" of Rasmussen) appeared normal. The vestibular membranous labyrinth, including sense organs and vestibular nerves, appeared normal throughout.

Cat 3. Behavioral audiograms revealed normal auditory thresholds to 8,000 cps with progressively increasing threshold losses for the frequencies 10,000, 12,000 and 14,000 cps. No response was made to frequencies above 14,000 cps at the maximum intensities available. The animal was anesthetized and an otoscopic examination performed to rule out the possibility of impacted cerumen as a cause for this hearing loss. The tympanic membrane appeared normal and there was no wax in the ear canal. Later an operation was performed on the apex of the cochlea. The animal died 24 hours postoperatively and was perfused immediately after death with 10 per cent formalin solution. As a result of the surgical procedure performed on the apex there was an acute destructive lesion involving the organ of Corti in the region from 16 mm. to the apex. In the basal coil from 0 to 6.5 mm. there was total atrophy of the organ of Corti and almost complete loss of spiral ganglion cells. There was decreased cellularity of the spiral ligament from 0 to 6.5 mm. region and the limbus from 3.5 to 6.5 mm. The stria vascularis was atrophic in the region from 2 to 4 mm. In the intermediate region from 6.5 mm. to 16 mm. there were changes in the organ of Corti consistent with a mild postoperative labyrinthitis so that the organ of Corti was not satisfactory for detailed morphological study. The efferent nerve bundle was partially degenerated in the lower basal turn but normal elsewhere. The vestibular membranous labyrinth, the sense organs and the vestibular nerves were normal throughout. There were no pathological changes in the blood vessels of the spiral ligament, stria vascularis or modiolus.

Cat 4. Cortical tests on this animal showed a threshold elevation for frequencies above 500 cps with a complete loss of response above 2,000 cps. Immediately after cortical testing the animal was sacrificed by intravital perfusion with Heidenhain-Susa solution. Histological examination showed advanced atrophic changes in all structures of the organ of Corti and almost complete loss of spiral ganglion cells to the 14 mm. region (see Fig. 3). There were less severe changes in the region from 14 to 18 mm. Decreased cellularity of the limbus extended from 3 to 17 mm. with a total loss of nuclei between 7 and 12 mm. The tectorial membrane was completely missing at the basal end, making its appear-

ance at 2 mm. and becoming normal at 5 mm. The spiral ligament appeared normal except for decreased cellularity in the region from 8.5 to 9.3 mm., and the stria vascularis was moderately atrophic in exactly the same region. Reissner's membrane was intact. The blood vessels of the

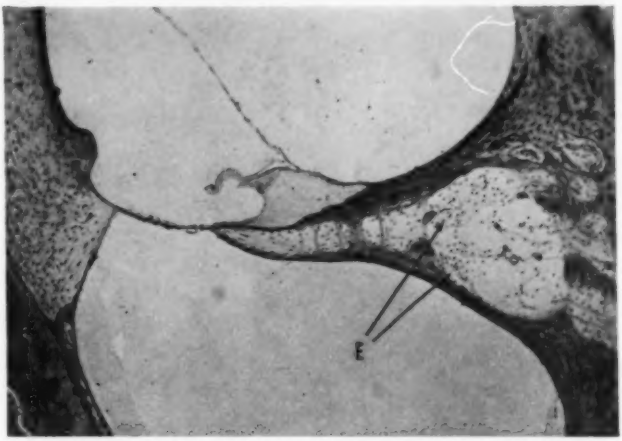

Fig. 3. Photomicrographs of the 11 mm. region of Cat 4. The organ of Corti is missing totally. The nuclei of the limbus are missing except for a few which remain near the vestibular lip. The spiral ganglion cells are missing except for four shrunken cells. The efferent nerve bundles ("E") have degenerated.

spiral ligament, stria vascularis, ganglion canal and modiolus appeared normal throughout. Many of the spiral ganglion cells which remained in the basal and middle turns were somewhat shrunken and had condensed nuclei. When compared with the normal appearing vestibular ganglion the contrast was distinct. The fibres of the efferent nerve bundles were degenerated up to the 17 mm. region, beyond which they appeared normal. The vestibular membranous labyrinth, including sense organs and vestibular nerves, appeared normal throughout.

* * *

The pathology in each of the four animals was much alike; the principal features were atrophic degenerative changes in the membranous cochlear labyrinth, including afferent and efferent nerve fibres, which began at the basal end and proceeded toward the apex (see Fig. 4).

The changes parallel closely the pathology described by Crowe, et al., for human ears with abrupt high tone deafness

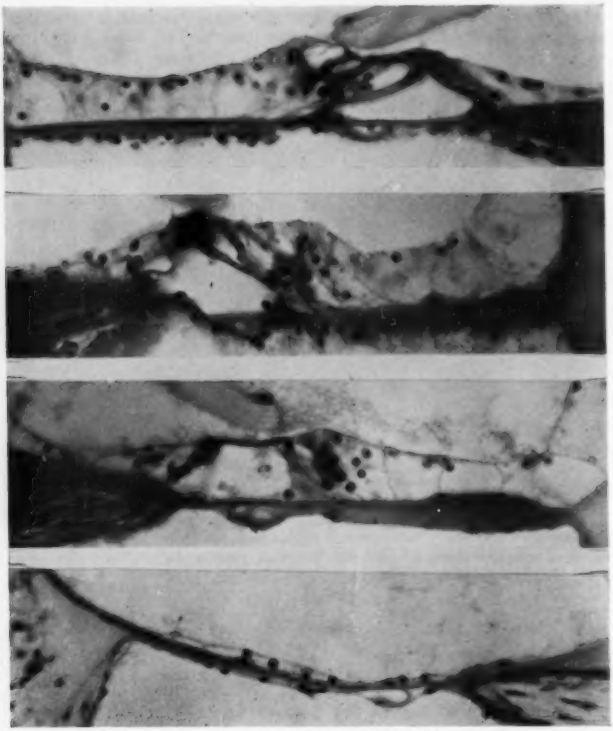

Fig. 4. Progressive stages of degeneration of the organ of Corti in the epithelial atrophy of presbycusis.

1. From the 18 mm. region of Cat 2. The hair cells and pillars are normal. The slight swelling of the external sulcus cells and slight flattening of Hensen's cells may be fixation artifact. The spiral ganglion was normal in this region.

2. From the 11 mm. region of Cat 2. A few outer hair cells are missing in this region. The inner pillar is crooked. The outer sulcus cells are swollen and Hensen's cells are slightly flattened. There was a loss of 10 per cent of the spiral ganglion cells.

3. From the 8 mm. region of Cat 2. The hair cells are missing. There is a total loss of pillars except for a remnant of the inner pillar. The sulcus cells and Hensen's cells are further degenerated. In this region 85 per cent of the spiral ganglion cells are missing.

4. From the 11 mm. region of Cat 4. The organ of Corti is missing entirely except for a few epithelial cells which lie on the basilar membrane. About 95 per cent of the spiral ganglion cells are missing in this region of the cochlea.

and is identical to that which I have seen in human ears in Dr. John Lindsay's collection at the University of Chicago.

The question arises whether the change originates in one of the structures with other elements suffering because of changes in the first. In this regard, it is known that the degenerative change could not be primary in either the hair cells or spiral ganglion, because either of these structures can be lost independently without imposing a degenerative change upon the other or upon the supporting elements.

The change could not be primary in the supporting elements, as I once proposed, because although this would explain the loss of hair cells and ganglion cells, it would not explain degeneration of the efferent nerve bundle. For example, in severe stimulation injury the efferent nerve bundle is known to persist even though the organ of Corti and its afferent nerve supply are completely lost.

It is also not likely that the atrophic changes in the membranous labyrinth and spiral ganglion are secondary to degeneration of the olivo-cochlear bundle in view of Rasmussen's experiments in which the bundle was cut without creating an atrophic change in the organ of Corti during a period of several weeks.

After a consideration of all the evidence it seems reasonable to assume that the degenerative change as it proceeds up the cochlear duct affects almost equally and simultaneously the various structures within it, including the afferent and efferent nerve fibres. The reason why the atrophic change selects the basal end is as great a mystery to me as the process of aging itself.

These cochlear changes probably are a manifestation of the same degenerative process affecting supporting tissues elsewhere in the body. The presbycusis curves for men and women show that it begins in middle age (see Fig. 5). A close corollary is to be found in the skin, another ectodermal structure. As early as the third decade of life the epidermis begins to lose its rete-peg pattern and becomes thinner. Beginning in the fourth decade there are changes in the dermis consisting of clumping and basophilic staining of collagen

fibres and hyalin degeneration and fragmentation of elastic fibres. These changes are responsible for the wrinkling and flaccidity of the skin of the aged.

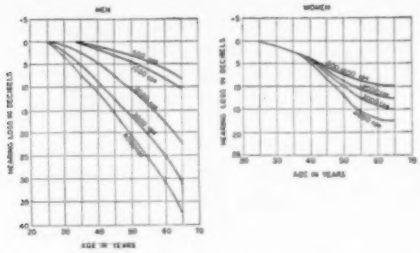

Fig. 5. Presbycusis curves for men and women, from "The Relations of Hearing Loss to Noise Exposure" Am. Standard Assn., 70 E. 49th St., N. Y. These curves were plotted from data obtained in large population surveys by Bunch, Steinberg, et al., and Webster, et al. In each of these studies the reference line (zero hearing loss) was the average hearing loss in the group between 20 and 29 years of age.

The pathological changes occurring in stimulation deafness[13,14] which differentiate it from epithelial atrophy are: *1.* The most severe injury is located some distance from the basal end of the cochlea (10 mm. in the cat) whereas in epithelial atrophy the changes are most advanced at the basal end; *2.* Loss of hair cells is always greater than loss of spiral ganglion cells, whereas in epithelial atrophy the loss of hair cells and spiral ganglion cells are closely parallel; *3.* The efferent nerve fibre bundles do not degenerate.

The pathological changes in non-suppurative labyrinthitis which differentiate it from epithelial atrophy are: *1.* It is more diffuse, usually involving the entire cochlea; *2.* Hair cell loss is greater than ganglion cell loss; *3.* There is histological evidence of inflammatory reaction, such as envelopment of tectorial membrane by epithelial cells.

In certain types of congenital deafness, such as that due to rubella, there are pathological changes in the saccule, as well as the cochlea, a situation which never exists in epithelial atrophy.

Structure	Magnitude of Injury
Hair Cells	Each hair cell rated as either normal, slight morphological change, severe morphological change, or missing.
Spiral Ganglion	Estimated percentage of ganglion cells missing, 0 to 100 per cent in 5 per cent steps.
Reissner's Membrane	Recorded as intact or torn.
Tectorial Membrane	Displacement not rated as abnormal. Structural abnormality rated from 0 to 100 per cent in 25 per cent steps.
Spiral Ligament Limbus	Each rated according to per cent of nuclei missing within structure from 0 to 100 per cent in 25 per cent steps.
Pillars Hensen's Cells Sulcus Cells	Each rated according to architectural appearance and number of cells remaining from 0 to 100 per cent in 25 per cent steps.
Stria Vascularis	Estimate of percentage atrophy, 0 to 100 per cent in 25 per cent steps.
Efferent Nerve Fibres	Estimate of percentage of fibres remaining 0 to 100 per cent in 25 per cent steps.

PRESBYCUSIS DUE TO NEURAL ATROPHY.

The second type of presbycusis I have termed neural atrophy because I believe it is due to a decrease in population of the neurons of the auditory nervous pathways. Ordinarily its onset is in older age, and is superimposed upon the epithelial atrophy already described. The pathological features are shown in the following case.

Mr. S., U. of Chicago. This 70-year-old carpenter developed carcinoma of the floor of the mouth with insurmountable cervical lymphatic metastases. When it became obvious that the ulcerating metastatic lesion was invading the common carotid artery the patient was admitted to the hospital for terminal care. After admission to the hospital an audiogram was partially accomplished for the right ear, four frequencies having been tested in consecutive order from 8,000 cps to 1,000 cps, when sudden profuse bleeding from the neck occurred, and the audiometric examination had to be discontinued (see Fig. 6). The patient was mentally alert and comfortable during the test, and the results were considered to be reliable by the audiologist.* The patient died five hours later. Thirty minutes after death the right ear drum was removed partially, the stapes was dislocated, and the middle ear was filled with 20 per cent formalin solution. The left ear was not injected because it was used as a control to determine the effectiveness of the injection method. The postmortem changes in this ear were severe so that it was not used in the present study. Three hours and 50 minutes after death the temporal bones were removed and placed in Helly's solution.

Histological examination revealed typical epithelial atrophy to the 3.3 mm. point. There were characteristic atrophic

* I am indebted to Mr. Duncan R. C. Scott for performing the audiogram.

changes in the pillars, stria vascularis, tectorial membrane, hair cells and ganglion cells to the 3.3 mm. region. Slight degenerative changes in the sulcus cells and Henson's cells extended to 3.6 mm. Reissner's membrane was torn in the region between 1 and 2.3 mm. No changes were seen in the blood vessels of the stria vascularis, spiral ligament or modiolus.

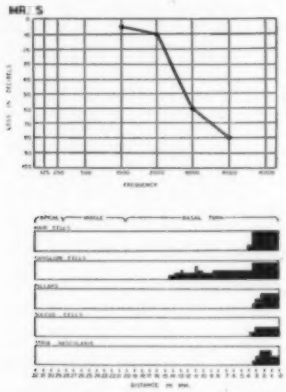

Fig. 6. Audiogram and cochlear chart of a 70-year-old man with high tone deafness. There is "epithelial atrophy" for 3½ mm. and a superimposed "neural atrophy" extending to 14.2 mm.

In addition there was 30 to 50 per cent loss of spiral ganglion cells extending from 3.3 to 14.2 mm. without accompanying change in the organ of Corti or other structures of the membranous labyrinth. It is more difficult to judge the percentage of ganglion cells missing in human than in animal ears, because the ganglion canal in the basal coil normally is not filled with neurons. Judgments for this ear were made by comparison with the ganglion cell density existing in normal ears of young persons. The efferent nerve bundle appeared normal throughout. The vestibular membranous labyrinth, including the sense organs and vestibular nerves, appeared normal. The ganglion cells which remained in the basal coil appeared normal.

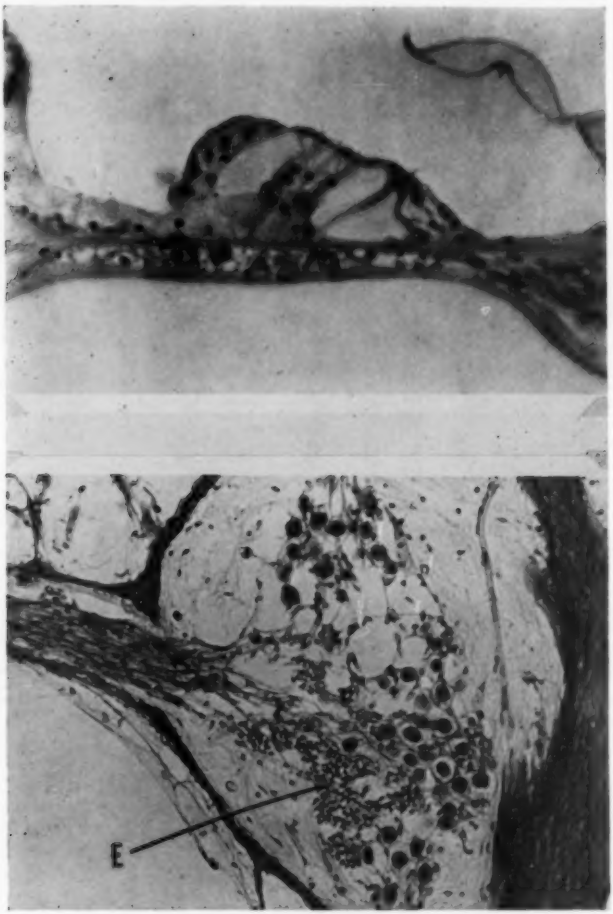

Fig. 7. Photomicrographs from the ear of Mr. S. Above: Normal organ of Corti from the 11.5 mm. region. Below: Spiral ganglion from the same region with an estimated loss of 30 per cent of the cells.

In the audiogram the frequencies have been arranged on a theoretical anatomical frequency scale. A similar scale was suggested by Koenig[15] and has been used for guinea pigs by Davis.[16] For the guinea pig frequencies above 1,000 cps were arranged logarithmically, and those below 1,000 cps arith-

metically, so that each 100 cycle interval was equal to the distance between 900 and 1,000 cps on the logarithmic scale. For the human, I have selected 31.25 cps as the lowest frequency of the audiogram and 20,000 cps as the highest; both extremes are an octave lower than that used for the cat. Justification for the low frequency limit is based on Bekesy's[17] observations that the lowest frequency still having a point of maximum displacement on the cochlear partition is about 25-35 cps. It is known that the high tone limit for young persons extends to 20,000 cps. The frequency of 500 cps was selected arbitrarily as a dividing point between the logarithmic and arithmetic scales.

For human ears, Crowe, et al., correlated hearing loss with location of nerve fibre lesions and thus related the 4,096 frequency to the 7-8 mm. region, and the 8,192 frequency with the 4-5 mm. region.[8] It should be pointed out, however, that correlations of this kind are complicated by the fact that there is not a close parallelism between nerve fibre population and pure tone threshold.[18]

A corollary to atrophy of the spiral ganglion exists in the brain, for it is well established that the aging brain suffers from a loss of nerve cells. The process develops independently of cerebral arteriosclerosis, but may co-exist with varying degrees of it. The clinical manifestations of loss of cortical neurons is senile dementia, characterized by a gradual diminution of physical and mental capacity, an exaggeration of prior personality traits, increasing intellectual failure, impairment of memory, errors of judgment and insomnia. The cell outlines of the cortical neurons become shrunken and irregular, the cytoplasm becomes hyperchromatic, the Nissl material decreases, the nuclei become basophilic and later there is glial proliferation and neuronophagia. Whereas senile dementia is a clinical manifestation of loss of cortical neurons, a loss of discrimination ability and auditory acuity are expressions of loss of neurons in the auditory pathways.

Neuron loss in the higher auditory centers also would be expected to create discrimination loss, and if severe enough, threshold losses, too. It may be that the threshold losses which Guild and Saxon could not explain on the basis of pathology

in the cochlea or spiral ganglion were due to neural degeneration at higher levels.

It is an established clinical observation that the deafness of aged patients is characterized by decreased auditory discrimination in the presence of disproportionately small loss in pure tone thresholds. This has been termed "phonemic regression." Experimental and clinical evidence has established that these relationships are characteristic of neural degeneration. For example, it has been shown experimentally that 75 percent of ganglion cells may be lost to certain regions of the cochlea without creating threshold elevations for frequencies having their fields of excitation in those regions, whereas greater ganglion cell losses do create threshold losses; furthermore, audiological studies on human patients with acoustic neurinomas have shown that decreased speech discrimination may exist with disproportionately small pure tone threshold losses.[18] The implication of this finding is that only a few nerve fibres are needed to carry impulses of threshold magnitude. I do not believe, therefore, that the spiral ganglion cell deficit in the 3.3 to 14.2 mm. regions can alone account for the threshold losses for the frequencies 4000 to 8000 cps. The most reasonable explanation, it seems to me, is that there are also deficits in the population of 2nd, 3rd and 4th order neurons and that the composite loss of neurons is sufficient to affect thresholds.

Along with other considerations it is important to establish the discrimination ability of the presbycusic patient when considering the usefulness of a hearing aid. The folly of amplifying markedly distorted patterns of auditory neural activity is well known. From the diagnostic standpoint it can be said that the neural atrophy type of presbycusis exists when the loss of discrimination ability is more severe than would be expected from limitation of high tone hearing alone.

The pathological anatomy existing in both the epithelial and neural types of presbycusis does not produce recruitment. It is known that recruitment requires an incomplete sensory lesion; in other words, there must be a partial loss of hair cells with normal or near-normal numbers of ganglion cells remaining within the fields of excitation.[19] Thus, at high intensities the hair cells which remain are capable of exciting

normal numbers of nerve fibers. This type of cochlear change commonly exists in toxic, inflammatory and traumatic lesions of the organ of Corti; therefore, the recruitment test can sometimes be of value in the differential diagnosis of high tone deafness in that its presence rules out presbycusis.

CONCLUSIONS.

A quantitative study was made of the pathological anatomy in the ears of several cats and humans with high tone deafness. The evidence indicates that there are two types of presbycusis. The first, termed "epithelial atrophy," is characterized by degenerative changes which begin at the basal end of the cochlear duct and proceed toward the apex, affecting almost equally and simultaneously the various structures within it, including the afferent and efferent nerve fibres. The process begins in middle age, progresses very slowly for decades, and manifests itself clinically by the familiar presbycusis curves. It is the otological manifestation of an aging process which affects all tissues, a close corollary existing in the skin where the changes are so characteristic they are used as a yardstick to measure the age of the individual.

The second type, termed "neural atrophy," is characterized by degeneration of spiral ganglion cells beginning at the basal end of the cochlea as well as neurons of the higher auditory pathways and is superimposed upon varying degrees of epithelial atrophy. Its onset is late in life, it progresses slowly for years, and is characterized clinically by high tone deafness with disproportionately severe loss in auditory discrimination. It is the otological manifestation of an aging process affecting the central nervous system which is characterized by a loss of neuron population and has a corollary in the cerebral cortex where loss of neurons results in senile dementia.

REFERENCES.

1. NEEDHAM, A. E.: Growth and Regeneration Rates in Relation to Age in the Crustacea. *Jour. Gerontol.*, 5:5-16, 1950.

2. BULLOUGH, W. S.: Age and Mitotic Activity in the Male Mouse, Mus Musculus L. *Jour. Exp. Biol.*, 26:261-286, 1949.

3. ANDREASEN, E.: Studies on Thymolymphatic System: Quantitative Investigations on Thymolymphatic System in Normal Rats at Different

Ages, Under Normal Conditions and During Inanition and Restitution After Starvation. *Acta Path. et Microbio., Scandinav. Supp.*, 49:1, 1943.

4. Simms, H. S., and Stolman, A.: Changes in Human Tissue Electrolytes in Senescence. *Science*, 86:269-270, 1937.

5. Lansing, A. F.: Cowdry's Problems of Aging. Williams & Wilkins, Baltimore, ed. 3, 1952.

6. Andrew, W.: Cellular Changes with Age. Chas. C. Thomas, Springfield, Ill. Publication 127 of American Lecture Series, 1952.

7. Zwaardemaker, H.: Der Verlust am Hohen Tonem mit Zunehmendem Alter. ein Neues Gesetz. *Arch. f. Ohrenh.*, 47:1899.

8. Crowe, S. J.; Guild, S. R., and Polvogt, L. M.: Observations on the Pathology of High Tone Deafness. *Bull. Johns Hopkins Hosp.*, 54:315-379, 1934.

9. Saxen, A.: Pathologie und Klinik Der Altersschwerhörigkeit. *Acta Otolaryngol. Supp.* 23, 1937; Inner Ear in Presbycusis. *Acta Otolaryngol.*, 41:213-227, 1952.

10. Schuknecht, H. F.: Techniques for Study of Cochlear Function and Pathology in Experimental Animals. *Arch. Otol.*, 58:377-397, 1953.

11. Hind, J. E., and Schuknecht, H. F.: A Cortical Test of Auditory Function in Experimentally Deafened Cats. *Jour Acoustic Soc. of Amer.*, 26:89-97, 1954.

12. Rasmussen, G. L.: Further Observations of the Efferent Cochlear Bundle. *Jour. Comp. Neurol.*, 99:61-74, Aug., 1953.

13. Lindquist, S.; Neff, W., and Schuknecht, H. F.: Stimulation Deafness: A Study of Hearing Losses Resulting from Exposure to Noise or to Blast Impulses. *Jour. Compar. and Physiolog. Psych.*, 47:406-411, 1954.

14. Schuknecht, H. F.; Neff, W. D., and Perlman, H. B.: An Experimental Study of Auditory Damage Following Blows to the Head. *Ann. Otol., Rhinol. and Laryngol.*, 60:273-289, 1951.

15. Koenig, W.: A New Frequency Scale for Acoustic Measurements. *Bell Laboratory Record*, p. 209, Aug., 1949.

16. Davis, H.; Gerandt, B. E.; Riesco-MacClure, J. S., and Covell, W. P.: Aural Microphonics in the Cochlea of the Guinea Pig. *Jour. Acous. Soc. Am.*, 21:502-510, 1949.

17. Bekesy, G. V., and Rosenblith, W. A.: The Mechanical Properties of the Ear. *Handbook of Experimental Psychology.* John Wiley & Sons., Inc., 107-115, 1951.

18. Schuknecht, H. F., and Woellner, R. C.: An Experimental and Clinical Study of Deafness from Lesions of the Cochlear Nerve. *Jour. Laryngol. and Otol.*, 69:75-97, Feb., 1955.

19. Schuknecht, H. F.: Lesions of the Organ of Corti. *Trans. Amer. Acad. Ophthalmol. and Otol.*, May-June, 1953.

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TRANS-SPHENOIDAL APPROACH TO THE PITUITARY GLAND.*

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The high mortality and technical difficulties of frontal craniotomy for the removal of pituitary tumors directed the surgeons (not the otolaryngologists) to the trans-sphenoidal approach to these lesions. This technique was first suggested by Schloffer[1] in 1906 and performed by him in 1907.[2] He employed a lateral incision for the dislocation of the external nose and popularized the procedure for the first decade of interest in this field.

It is reported that more than 100 operations of this type were performed by various surgeons during this early period. Von Eiselberg[3] used this method in performing the first operation for Frolich's syndrome in 1907, and Hochenegg[4] performed the first operation for acromegalia in 1908. Different methods of dislocating the external nose in an effort to reach the sphenoidal sinus were attempted by a number of surgeons. Dislocation from above downward was used by Proust[5] in 1908; from below upward by Kanavel in 1910,[6] and again by Kanavel and Grinker in the same year (1910).[7]

A sublabial incision was devised by Halstead in 1910.[8] Hirsch,[9] in 1909, suggested the trans-sphenoidal endonasal operation with local anesthesia and performed it successfully in 1910. This procedure was first made possible by opening one sphenoidal cavity through the ethmoidal route by Hajek.[10] From observations derived from this method, Hirsch developed the septal approach to both sphenoidal sinuses by performing

* Read at the Fifty-Ninth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Hollywood, Fla., March 17, 1955.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication March 23, 1955.

a submucous resection of the nasal septum.[11] Cushing subsequently combined the sublabial incision of Halstead with the septal method of Hirsch in 1912.[12] An interest in this surgical procedure became manifest among rhinologists of this period, and various surgical approaches were devised and recommended for operations upon the pituitary gland. The paranasal method of Chiari in 1912[13] using the external ethmoidectomy was employed. The gingivo-labial incision and trans-maxillary sinus route of Fein[14] and Denker[15] and the palatine methods of Preysing[16] and Tiefenthal[17] enjoyed a brief period of popularity. It soon became obvious, however, that none of these methods offered the advantages of the septal procedure, especially in regard to postoperative management and secondary operations for recurrences.

It would seem that almost everything that could be written about chromophobe pituitary adenomas has been published, but recent endocrine studies furnish fresh information which necessarily alters some of our concepts in respect to diagnosis and treatment. There can be little question that this new knowledge gives us a better understanding of the effects of these lesions upon the general economy of the patient, and points the way to improved management and a more hopeful prognosis.

Basic studies of pituitary gland secretion have established the fact that when the anterior lobe of a normal gland has been removed, there is an abrupt interruption in the production of four important hormones with a simultaneous loss of the stimulating effects which each one has upon specific "target organs."

1. *The somatotropic or growth hormone,* whose target organ is protein tissue (muscle, bone, etc.). When lost, the individual's growth is interrupted, stature remains small, and his appearance is that of a younger individual than his chronological age. X-rays of the skeletal framework may demonstrate delayed bone growth.

2. *Loss of the gonadotropic hormone,* the follicle stimulating hormone, results in subnormal function of the ovaries

in the female and the testes in the male. In the former, one will note a delayed appearance or disappearance of the secondary sexual characteristics and an amenorrhea. The skin is smooth and there is an absence or decrease of pubic and axillary hair. Breast development is decreased or absent. In the male, one likewise observes the smooth "baby" skin and scant or absent pubic and axillary hair. The penis, scrotum and testicles are under-developed and there is a delayed appearance or decrease of facial beard.

3. The thyrotropic hormone stimulates the thyroid gland. Its loss is characterized by decreased sweating, ease of fatigue, loss of stamina, and somnolence. One will note a dry to cool skin, bradycardia, loss of body hair, and slow responses.

4. The last important hormone, *adrenocorticotropic hormone* (ACTH) stimulates the adrenal cortex. Its absence is characterized by a hypoglycemia, postural hypotension, and decreased strength and stamina. One may also note a pale, smooth skin (alabaster skin), decreased axillary hair and decreased skin pigmentation. With the loss of the four essential hormones of the pituitary gland, certain important urinary findings are noted.

The growth of a chromophobe tumor is usually slow and obviously not attended by an abrupt interruption of anterior lobe secretions. Thus we may observe a variety of target organ deficiencies with emphasis upon one or two of them, or a combination of all four. It is of clinical significance that target organ deficiency usually appears in a selective sequence and in accordance with nature's design. The tropic hormones, least important to life, are the first to be lost; while the most vital for survival, are the last to disappear. Thus we observe that growth and gonadotropic hormones are the first to be depleted, while thyrotropic and adrenotropic hormones are the final ones which fail to function. It is, of course, obvious that one cannot determine the presence or absence of the growth hormone in the adult nor can one observe clinically the status of the gonadotropic sphere in the period preceding puberty.

CLINICAL OBSERVATIONS.

The chief complaints of the patients with chromophobe adenoma of the pituitary, who have come to our attention, have been blurred vision and headaches. As the lesion encroaches upon the visual pathways, one usually finds a right or a left homononymous hemianopsia due to pressure upon one of the optic tracts. There may be only a slight temporal defect of the visual field on one side or the other. The classical bi-temporal hemianopsia was observed in four of twelve cases of chromophobe adenoma recently studied by us. Quoting from our textbooks it is observed that this typical visual finding occurs in only 50 per cent of the cases. At the first examination we frequently find that the patient is so blind that the type of hemianopsia which previously existed cannot be discovered.

Associated with a blurring of vision, one may observe a partial or complete paralysis of the external ocular muscles, chiefly those supplied by the third cranial nerve.[11] I should like to emphasize that a pale disc does not necessarily indicate an alarming degree of optic atrophy. In several of our patients the pallor was extreme, but an amazing amount of visual acuity returned when pressure was removed from the visual pathways by surgical interference. Walker and Cushing,[10] observed in histological examinations of optic nerves of this character, that the amount of fibers preserved was often substantially more than expected. One is frequently gratified to observe a remarkable improvement in vision after surgical procedures for chromophobe adenomas in patients who have been doomed to an optic atrophy of an irreversible character.

Headache may be the first symptom of which the patient complains. It may be excruciatingly severe. At first it is usually bi-temporal but later tends to become more or less a general type. An early symptom in the male is loss of libido and in the female a dimunition or cessation of menstruation. Body hairs become scant and some degree of obesity is often in evidence. A subnormal basal metabolic rate is a frequent finding. The signs and symptoms change in different patients from time to time depending upon the size of the lesion, its proportions in relation to neighboring structures, and its

pathological behavior. The roentgenographic examination is the most important method of determining the size and anatomical relationships of the pituitary adenoma. In the Roentgenogram, one may note merely an enlargement of the sella or a deformity of its floor, and finally perhaps complete destruction of the sella turcica.

Cerebral angiography is one of the most important means of demonstrating the intracranial extension of the lesion and displacement of the internal carotid arteries. It is highly essential to visualize the carotid system in this manner before surgical intervention is instituted. The relationship of the internal carotid arteries to the lesion is an observation that must be made in the interest of the patient's safety before the trans-sphenoidal approach to the adenoma is attempted.

It is often possible to speculate with some degree of accuracy upon the nature of the lesion by evaluating its clinical behavior. For example, rapid growth, as determined by the rapidity of symptoms and extensive destruction of the base of the skull observed in the Roentgenogram, suggests the presence of an adeno-carcinoma. Dramatic clinical symptoms such as sudden blindness, extra-ocular palsies, coma, signs of intracranial and subarytenoid hemorrhage, and acute pan-hypopituitarism have an important clinical significance in determining when and how the patient should be treated. As a rule, signs and symptoms of this character call for an emergency surgical attack upon the lesion if the patient's vision or perhaps his life is to be saved. Here again, angiography may be most useful in differentiating an adenoma of the pituitary gland from a bleeding intracranial aneurism.

MANAGEMENT.

Because the incidence of pituitary adenomas is relatively low the number seen in any one institution is usually not great. It is encumbent upon us, therefore, to accumulate and evaluate the experiences of other qualified workers in this field and to draw heavily from the concepts and philosophy of treatment of such outstanding contributors as H. Dubney Kerr,[20] Franz Buschke,[21] L. M. Davidoff,[22] C. C. Dyke,[23] J. E. Paterson,[24] M. C. Sosman,[25] and others.

TABLE I. CLINICAL MANIFESTATIONS OF TARGET ORGAN DEFICIENCY.

Trophic Hormone	Target Organ	Symptoms	Signs	Laboratory Data
Somatotrophic (STH) or Growth (GH)	Protein tissue (muscle, bone, etc.)	"Stopped growing"	Short stature Appears younger than chronological age	X-rays of wrists show delayed bone growth
Gonadotrophic: (Follicle Stimulating Hormone—FSH—only one which can be assayed)	Ovaries	Delayed appearance or disappearance of secondary sexual characteristics. Amenorrhea	Smooth "baby" skin Absence or decrease of pubic and axillary hair Absence or decrease of breast development	Low urinary excretion of: Follicle stimulating hormone (FSH) Estrogens 17-ketosteroids
	Testes		Smooth "baby" skin Absence or decrease of pubic, axillary and scrotal development Absence or decrease of penile, testicular and scrotal development Delayed appearance or decrease of facial beard	Low urinary excretion of: 17-ketosteroids
Thyrotrophic (TSH)	Thyroid	Cold intolerance Decreased sweating Ease of fatigue Loss of stamina Somnolence	Dry, cool skin Bradycardia Coarse hair Loss of body hair Slow responses	Low BMR High (or normal) cholesterol Low protein bound iodine (PHI) Low I_{131} uptake
Adrenocorticotrophic (ACTH)	Adrenal Cortex	Pre-breakfast hypoglycemia (weakness, headache relieved by food)	Pale, smooth skin ("alabaster skin") Decreased axillary hair	Low FBS Low Na and Cl (occasionally)
		Postural hypotension (faintness on standing)	Postural hypotension (fall in systolic and diastolic BP on standing)	Low 17-hydroxysteroids (17-OH) Low 17-ketosteroids (17-KS)
		Decreased strength and stamina Decreased skin pigmentation Loss of axillary and pubic hair		

If the diagnosis can be established clinically at an early date and with a fair degree of accuracy, it is our opinion that irradiation should be used as the primary treatment. This is accomplished by a single course technique which consists of delivering a tumor dose of about 3000 r in three weeks through two lateral fields. It has been definitely shown that this order of dosage is sufficient to arrest permanently the growth of such a lesion in some patients. Our Roentgenologist reports that this technique will give satisfactory results in 60 to 70 per cent of his cases; however, the patients coming to my attention are those who have not responded satisfactorily to X-ray therapy. They come to me with the signs and symptoms of advanced growth which frequently calls for emergency surgical measures. The indications for operation may be listed as follows:

1. The imminent possibility of blindness or rapidly decreasing visual fields.

2. Severe intractable headache not relieved by irradiation.

3. Cystic changes suggestive of long standing disease, or a very large lesion upon which X-ray therapy would probably have little effect.

4. Evidence of further growth of the adenoma after a course of irradiation of the dosage listed above. (Additional irradiation would be hazardous.)

5. An indefinite diagnosis clinically which calls for an exploratory operation. (If a pituitary adenoma is found, post-operative irradiation may be employed.)

ENDOCRINE MANAGEMENT.

The preparation of the patient for operation calls for the administration of exogenous adrenal steroid compounds for the following reasons:

1. Unless time-consuming tests for adreno-cortical reserve are done, one cannot be certain that a debilitated pituitary adrenal axis will be capable of responding adequately to the stress of a surgical procedure.

2. In order to obtain adequate removal of the adenoma, it may occasionally be necessary to eviscerate the contents of the sella turcica including the remnant of functioning adeno-

hypophysis. Adreno-cortical tropic secretion would cease as of that moment and adreno-cortical collapse could be predicted within 24 hours. In past experience, the importance of this phenomenon was not realized and some of our unexplained deaths after hypophysial operations were undoubtedly caused by acute hypopituitarism.

3. No untoward complications due to steroid therapy have been observed in patients receiving cortisone before, during, and after surgical interference. Of equal importance, also, is the maintenance of adequate electrolyte balance.

In our clinic, cortisone (compound E) has been used as the agent of choice, although compound *F* may ultimately replace cortisone when an intra-muscular preparation becomes available.

The administration of active adreno-cortical compound is preferred to the use of adreno-cortical tropic hormone because the response to intra-muscular administered ACTH, either the aqueous or the gel preparation, is not predictable. The rate of absorption or local inactivation of the agent in the muscle is variable. (See Table II.)

TABLE II.

PRE-OPERATIVE AND POST-OPERATIVE CORTISONE SCHEDULE.

Day	Milligrams of Cortisone (Intramuscular) According to the Weight of the Patient		
	30-40 lbs.	70-90 lbs.	130 lbs. or more
48 or 36 hours pre-operatively	75	100	200
24 hours pre-operatively	75	100	200
6 A. M. day of operation*	50	50	100
Post-operatively, first two days	25 q. 8 hours	25 q. 6 hours	50 q. 6 hours
3rd P. O. day†	25 q. 12 hours	25 q. 8 hours	50 q. 8 hours
4th P. O. day	25	25 q. 12 hours‡	50 q. 12 hours‡
5th P. O. day	12½	25	50
6th P. O. day	12½	12½	25

* Hydrocortisone (Compound F) is also kept on hand in the operating room for emergency intravenous administration (25 mg. stat, and 10 mg. per hour by continuous drip thereafter) in case of sudden peripheral vascular collapse. This has never occurred, however, in a patient who was prepared for operation with cortisone according to the above schedule.

† The rapidity with which cortisone administration is tapered off depends upon the speed of convalescence of the patient.

‡ When the patient is taking food, cortisone may be given orally instead of intramuscularly, in divided doses throughout the day.

TECHNIQUE OF OPERATION.

There is no need here for a detailed description of the submucous resection of the septum. There are a few maneuvers perhaps about which one might quibble, but on the whole our objective is to perform a satisfactory septum operation, control bleeding, and obtain a good view of the rostrum of the sphenoid. The latter structure which forms the anterior wall of the sphenoidal sinus is removed as extensively as possible in order to permit a good view of the sinus cavity. At this stage it is often discovered that the floor of the sella has been eroded and that the tumor is presenting within the sphenoidal sinus. Its capsule is then incised and the contents of the lesion removed with curettage and suction. This can be accomplished with considerable ease when the lesion is truly the chromophobe type and growing downward into the sinus cavity. If the floor of the sella is intact, this must be removed with chisel and biting forceps to expose the capsule of the tumor so that the latter may be incised and contents evacuated. Every effort should be made to remove the anterior and inferior circumference of the capsule as extensively as possible, care being taken to avoid the lateral walls which lie in intimate relationship to the internal carotid arteries.

It is to be emphasized that one can usually remove as much of the capsule of the tumor through a trans-sphenoidal exposure as can be accomplished from above by turning a frontotemporal flap. The neurosurgeon does not attempt to peel the capsule of a chromophobe adenoma off the internal carotid arteries and the cavernous sinuses nor should the rhinologist undertake such an extensive procedure by his method of attack. This is not necessary. The accomplishments in terms of amount of capsule removed are approximately the same by both methods, and obviously much less formidable and hazardous when the intranasal route is employed. It is desirable to expose the sphenoidal sinus widely and to drain it into one of the nasal cavities through a vertical incision in the posterior extremity of one of the septal flaps. A Penrose drain introduced through the nares into the sphenoidal cavity may be employed for two or three days after the operation. The septal flaps are then approximated by any of the numerous

methods which the rhinologist has been accustomed to employ. Fortunately, pre-operative preparation of the patient with antibiotics and chemotherapeutic agents and post-operative support by their continued administration now allay our fears of the serious complications which frustrated our efforts to use this operation successfully in the early decades of the century.

CONCLUSIONS.

1. The chromophobe adenoma of the pituitary can produce a variety of signs and symptoms by pressure upon neighboring structures and disturbances of endocrine functions of the gland. The common initial and obvious clinical symptoms are headache and blurred vision.

2. The radiographic examination of the skull usually furnishes the final clue to the identity of the lesion. In this connection angiography is of utmost importance in determining the size of the adenoma and position of the internal carotid arteries.

3. Recent scientific investigations of the functions of the pituitary adrenal axis furnish us new and important knowledge referable to the pre-operative and post-operative endocrine management of the patient.

4. It is generally conceded that the trans-sphenoidal approach to the chromophobe tumor can accomplish its resection as effectively as the more hazardous procedure of turning an osteoplastic flap in the fronto-temporal region. The adenoma is usually soft, cystic in architecture, with a brownish syrupy contents which can be removed by curettage and suction. Neither the neuro-surgeon nor the rhinologist should attempt complete eradication of the lesion. Our experience with X-ray therapy has been discouraging in that many patients who have been exposed to this form of therapy have eventually come to operation.

5. The effects of the operation are often spectacular. Vision frequently returns several hours later, and the headaches are often relieved before the patient is returned to his room. It goes without saying that the patient must invariably have the protection of the antibiotics and possibly also the chemotherapeutic agents against post-operative meningitis.

BIBLIOGRAPHY.

1. SCHLOFFER, H.: Zur Frage der Operationen an der Hypophyse. *Beitr. klin. Chir.*, 50:767, 1906.

2. SCHLOFFER, H.: Erfolgreiche Operation eines Hypophysentumors auf naselem Wege. *Wien. klin. Wchnschr.*, 20:621, 1075, 1907.

3. VON EISELBERG, A.: Die Hypophyse, Vienna, Urban & Schwarzenberg, pp. 34-38, 1930.

4. HOCHENEGG, J.: Zur Therapie von Hypophsentumoren. *Deutsche Ztschr. Chir.*, 100:317, 1909.

5. PROUST, R.: Chirurgie de l'Hypophse. *Jour. Chir.*, 1:668, 1908.

6. KANAVEL, A. B.: Removal of Tumors of the Pituitary Body by an Infranasal Route. *J. A. M. A.*, 53:1704, Nov. 20, 1909.

7. KANAVEL, A. B., and GRINKER, J.: Removal of Tumors of the Pituitary Body. *Surg., Gynec. and Obst.*, 10:414, 1910.

8. HALSTEAD, A. E.: Remarks on the Operative Treatment of Tumors of the Hypophysis: Report of Two Cases Operated on by an Oro-Nasal Method. *Surg., Gynec. and Obst.*, 10:494, 1910.

9. HIRSCH, O.: Eine neue Methode den endonaselen Operation von Hypophysentumoren. *Wien med. Wchnschr.*, 59:636, 1909.

10. HAJEK, M.: Zur Diagnose und Intranaselen Chirurgischen Behandlung des Hinteren Siebeinlabyrinthes. *Arch f. Laryngol.*, 16:105, 1904.

11. HIRSCH, O.: Über Methoden der Operativen Behandlung von Hypophysentumoren auf Endonaselem Wege. *Arch Laryngol.*, 24:129. 1910. Symptoms and Treatment of Pituitary Tumors. A. M. A. *Arch Otolaryngol.*, 55:268, 1952.

12. CUSHING, H.: The Pituitary and Its Disorders. J. B. Lippencott Company, Philadelphia, 1912, p. 300.

13. CHIARI, O.: Über eine Modification der Schloffer'schen Operation von Tumoren der Hypophyse. *Wien. klin. Wchnschr.*, 25:5, 1912.

14. FEIN, J.: Zur Operation der Hypophyse. *Wien. klin. Wchnschr.*, 23:1035, 1910.

15. DENKER, A.: Drei Fälle Operiert nach Transmaxillarer Methode. *Internat. Zentralbl. Laryngol.*, 37:225, 1921.

16. PREYSING, H.: Beitrage zur Operation der Hypophyse. *Verhandl. Deutsch. Laryngol.*, 20:51, 1913.

17. TIEFENTHAL, G.: Technik der Hypophysenoperation. *Munchener med. Wchnschr.*, 67:794, 1920.

18. LIST, C. F.: Management of Chromophobe Pituitary Adenoma. *Neurology*, 3:825, 1953.

19. WALKER, C. B., and CUSHING, H.: Distortions of the Visual Fields in Cases of Brain Tumors. *Arch Ophth.*, 47:119, 1918.

20. KERR, H. D.: Irradiation of Pituitary Tumors. *Am. Jour. Roentgen.*, 60:348, 1948.

21. BUSCHKE, F.: Radiotherapy of Pituitary Adenomas. *West. Jour. Surg., Obs. and Gynec.*, 58:271, 1950.

22. DAVIDOFF, L. M., and FEIRING, E. H.: Surgical Treatment of the Pituitary Body. *Am. Jour. Surg.*, 75:99, 1943.

23. DYKE, C. G., and HARE, C. C.: Roentgen Therapy of Pituitary Tumors: Report of 63 cases. *Assoc. Research. Nerv. and Men. Dis.*, Proc. 17:651, 1938.

24. PATERSON, J. E.: Cystic Pituitary Adenomata. *Jour. Neur., Neurosurg., and Psych.*, 11:280, 1948.

25. SOSMAN, M. C.: Irradiation in the Treatment of Pituitary Adenomas. *Proceedings of the Interstate Post-Graduate Medical Assembly of North America*, St. Louis, Oct. 18, 1937.

EPISTAXIS—EVALUATION OF SURGICAL CARE.*

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In 1947, at the conclusion of my resident training program, I had occasion to be associated in the care of a physician with epistaxis. During the first week of hospitalization he had recurring bleeding in spite of anterior and posterior nasal packing under anesthesia. He went on to develop the usual anxiety over his predicament, and inspired the development of a surgical approach for ligation which was effective, quickly curative, non-disabling, and surgically sound.

His individual reaction the day after surgery, comfortable, without bleeding or nasal packing, was: "Why the hell didn't you do this in the first place?" Fortunately, most cases of epistaxis do not require surgical ligation for definitive therapy; however, the question arises: is our conservatism and reluctance to use surgical therapy justified; and, if so, when do we draw the line and intervene?

An analysis of the medical literature and hospital records came subsequent to this event, and with it the stimulus to create a program for definitive care of serious epistaxis in accord with accepted basic surgical principles.

Spontaneous hemorrhage from the nose is a frequent occurrence in the practice of medicine. In the vast majority of these patients the arrest of hemorrhage is either spontaneous, self-controlled, or easily managed by local therapy at the office. Such local therapy takes the form of electro-coagulation, topical application of coagulants and escharotics, pressure packing of various forms, and many other methods advocated by individual reports in the literature.[1-5]

In the main these methods are equally effective, if not equally efficient in controlling hemorrhage, because the point

*Read as Candidate's Thesis, at the Fifty-ninth Annual Meeting of the American Laryngological, Rhinological, and Otological Society, Inc., Hollywood, Fla., March 17, 1955.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, March 23, 1955.

of bleeding is visible and can be exposed to direct local attack by any of the therapeutic procedures. There remains, however, a group of cases where bleeding is persistent and recurrent, and the loss of blood is so profuse that a serious state of anemia and shock with possible death ensues. To these cases we have affixed the diagnosis of serious epistaxis, for they create a problem demanding serious consideration as to therapy and cause great anxiety to all individuals concerned.

For the purpose of this discussion, serious epistaxis refers to those patients having persistent nasal bleeding who, because of the excessive loss of blood, require preliminary emergency anterior and posterior nasal packing for the control of bleeding and hospitalization for definitive treatment; it does not include cases of anterior bleeding which can be localized and easily controlled by coagulation or localized pressure by spot packing.

Modern textbooks in otorhinolaryngology devote little consideration to the subject of serious epistaxis and the management of this problem, leaving one with the impression that this condition does not occur so frequently as a review of the literature would indicate. Ogura and Senturia,[6] reporting in 1949, cited their investigation of 130 cases requiring hospitalization over a 15-year period; these were not all cases of severe bleeding as defined within the limits of this paper. Hallberg,[7] reporting from the records of the Mayo Clinic, cited 212 cases over a 20-year span. At the Eye and Ear Hospital, University of Pittsburgh, uncontrollable epistaxis requiring packing and hospitalization accounted for 235 admissions in the five-year period from 1947 to 1951, or an average of 47 cases per year; of these, 10 required surgical intervention other than packing. This admission rate is comparable to the admission rate for other common otorhinolaryngological procedures. The average hospital stay of the patients was 10 to 12 days, thus surpassing comparative figures for most other conditions requiring hospitalization in otorhinolaryngology.

It is not the purpose of this analysis to discuss the problem of epistaxis from the standpoint of etiologic factors. In the specialty of rhinology, serious epistaxis must be accepted as a primary condition requiring specific therapy. The treatment

of the causative diseases which have epistaxis as an associated symptom is not considered when developing a therapeutic regime for the immediate management of these cases.

It is commonly agreed that serious epistaxis is a problem of advancing years, occurring with increasing severity and frequency in individuals over 45 years of age. The pathologic changes in the blood vessel walls of the aged, resulting in fibrosis with loss of their inherent ability to control bleeding by retraction and contraction, account for the high percentage of cases recognized as being secondary to hypertensive and arteriosclerotic vascular changes.

Another factor responsible for spontaneous arrest of hemorrhage is clotting due to the end reaction of thrombin and thromboplastin; unfortunately, this alone is often inadequate for spontaneous control of bleeding from vascular channels the size of those involved in severe epistaxis. Blood dyscrasias, inflammations, systemic diseases, neoplasms, organic nasal disorders, and trauma are other common diseases which frequently have associated epistaxis. They are of secondary importance, however, in the treatment of this emergency problem.

Reporters to the medical literature over a period of years have been in agreement on the academic questions of etiology, incidence, and anatomy. The morbidity and mortality associated with epistaxis have been recognized. There is disagreement, however, on a definitive plan of therapy for effective and efficient permanent control of severe bleeding from the nose, except that "ligation may be a life-saving procedure." [5-10]

The relationship of the vascular supply to the surgical landmarks of the interior of the nose is of importance in determining the means of controlling bleeding by surgical intervention.

Severe epistaxis is associated with bleeding from the anterior ethmoid or sphenopalatine arteries. While the latter artery is the predominant of the two, there is an intimate anastomosis of their septal and lateral nasal branches. They are terminal branches of the external and internal divisions of

the carotid system. The external carotid artery enters the nose as the sphenopalatine division of the internal maxillary artery and supplies the posterior and inferior two-thirds of the medial and lateral wall of the nose. On emerging from the sphenopalatine foramen in the middle meatus, it then crosses in the mucosa of the anterior inferior border of the sphenoid sinus and separates into a lateral nasal and medial septal division.

The lateral branch descends to supply the mucosa over the inferior turbinate and its meatus. The vessel anastomoses with the nasal branches of the descending palatine artery. The medial division of the artery terminates on the septum by anastomosing with the ethmoidal arteries and the septal divisions of the superior labial artery from the external maxillary portion of the external carotid artery. The internal carotid system terminates in the nose as the anterior and posterior ethmoidal vessels; the latter arteries are of small caliber and, like the septal branches of the external maxillary, are unimportant except for the anastomotic pathways they contribute. The anterior ethmoidal artery takes origin in the orbit from the opthalmic artery and enters the nasal space from the medial wall of the orbit. It enters the nose from the crista galli and supplies the mucous membranes lining the sinus spaces above the middle turbinate and also gives off meningeal branches to the dura.

The middle turbinate is the anatomical landmark dividing these two main arterial systems. Bleeding from the anterior ethmoid artery is anterior and superior to the middle turbinate, while bleeding from the sphenopalatine is located posterior and inferior to the turbinate.

The carotid system is unique in regard to its multiple pathways of anastomosis. The internal carotid artery, which is free of branches in the neck, has a free anastomosis at the Circle of Willis with the corresponding vessel of the opposite side. The capability of this anastomosis to maintain cerebral blood flow was demonstrated by the experimental work of Shenkin[11] and his co-workers. Dandy[12] and Sweet,[13] reporting on changes in pressure associated with carotid ligation, also

verified the ability of this collateral circulation to develop and maintain pressure.

Matson and Woodhall,[14] investigating the effects of ligation of the internal carotid artery, proximal or distal to the cavernous sinus, failed to produce blindness through thrombosis

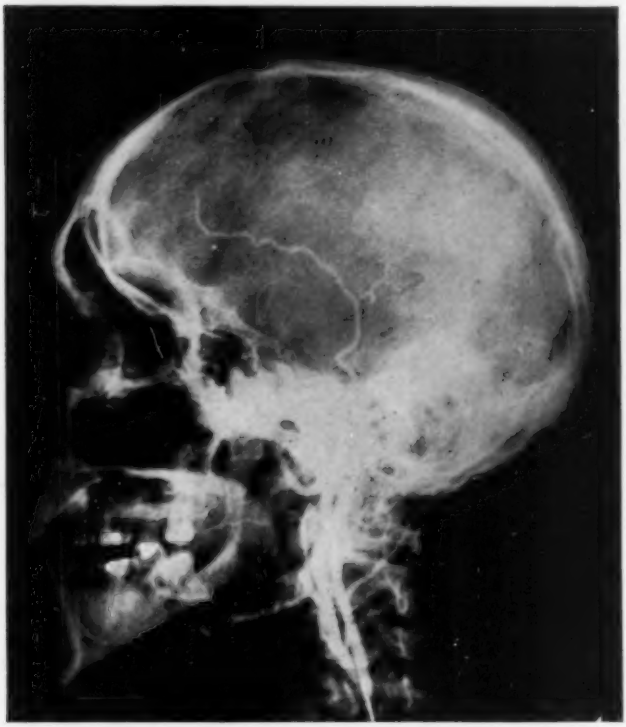

Fig. 1. Arteriography of the carotid system demonstrating the multiple, closely related, anastomotic pathways. Lateral view.

of the ophthalmic artery. This was offered as clinical proof of the rich anastomosis between the ophthalmic artery and orbital branches of the external carotid artery. This was confirmed by studies of Walsh and King.[15]

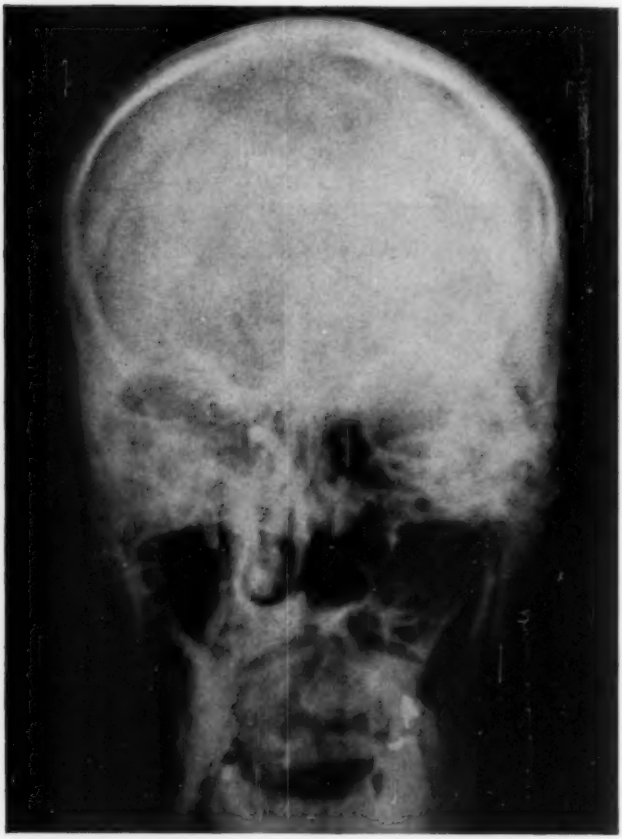

Fig. 2. Arteriography of the carotid system demonstrating the multiple, closely related, anastomotic pathways. Anterior-posterior view.

The external carotid artery has nine main branches and many secondary branches between its origin from the common carotid artery and the termination in the nose as the sphenopalatine artery. The anastomotic pathways between these various branches and their corresponding branches of the opposite side have been described anatomically[16] and demonstrated surgically. Wiley and Sugarbaker[17] reported on the

ineffectiveness of unilateral external carotid ligation for control of bleeding in cases of malignancy about the maxilla; they demonstrated the effectiveness of bilateral temporary or "tourniquet" ligation of these vessels. The significance of this vast anastomotic pattern has been demonstrated by the neurosurgeons and by arteriography of the carotid system[18] (see Figs. 1 and 2).

The control of hemorrhage is a basic principle which has been recognized and accepted from the beginning of surgery. Permanent control of hemorrhage is obtained by cauterization, pressure, or ligation.[19]

The efficiency of cauterization by thermocautery is dependent on the searing of tissues. This procedure is satisfactory in controlling hemorrhage in cases of simple epistaxis but is unreliable where bleeding is from larger arteries, generally invisible because of their posterior position in the nose.

In general, surgical control of bleeding is obtained through use of a hemostat, while in rhinology pressure control of bleeding is usually dependent on packing. The effectiveness of nasal packing is dependent upon spot pressure at the bleeding site when it is visible or spot packing over the proximal portion of the bleeding vessel in all other cases. Packing must, therefore, be directed between the middle turbinate and septum and laterally over the anterior wall of the sphenoid sinus toward the sphenopalatine foramen.

Such pressure is best obtained by using one-half inch mesh gauze, commonly referred to as mastoid packing, and placing it in this position with a Freer submucous elevator. If properly inserted this should immediately control all nasal bleeding secondary to sphenopalatine artery rupture. When bleeding is uncontrolled the packing should be continued forward to the anterior and superior portion of the middle turbinate so as to compress ethmoidal vessels and anastomotic channels of the ethmoid and sphenopalatine arteries.

The effectiveness of this packing is dependent on the precision of its insertion and the firmness of pressure. It is most efficiently inserted under anesthesia with a posterior plug in place. One cannot accomplish this end result in a restless,

uncooperative, anxious individual without anesthesia. Failure to control bleeding with this procedure is generally due to incomplete removal of blood clots from the nasal space prior to insertion of the packing or inefficient, loose packing which permits continuous oozing and clotting of blood in pockets between the gauze.

Removal of the packing is accomplished in multiple stages. After 72 hours a portion of the packing is withdrawn; the amount removed is usually indicated by the first appearance of blood staining on the gauze. This process is repeated daily until all the packing has been removed. Vascular ligation is reserved for those cases where bleeding has recurred in spite of precision packing. Ligation should be resorted to when continued bleeding endangers the patient's life due to progressive ischemia, lowered blood pressure, tachycardia, cardiac insufficiency, and coronary occlusion.

Ligation is a permanent curative procedure only when performed according to the accepted surgical principle of terminal ligation of vessels for control of hemorrhage. In the historical analysis of ligation for the control of epistaxis there were advocated procedures which failed to conform to this principle. The earliest surgical approach was that of common carotid artery ligation as advocated by Abernathy[20] in the 18th century. It is commonly accepted today, in spite of occasional reports in the literature, that common carotid artery ligation is unjustified because of its inefficiency and high mortality rate.

Ligation of the common carotid artery will restrict only 50 per cent of the flow of blood through the internal carotid artery. The ethmoidal and sphenopalatine circulation will be unaffected because of the adequacy of the Circle of Willis and its collateral circulation. The mortality rate for this procedure is expressed by various authors[22-23] as 15 per cent or higher because of the sudden closure of cerebral circulation in patients already anemic and undergoing cardio-vascular stress.

Internal carotid artery ligation is a more dangerous and undesirable procedure than common carotid artery ligation.

Voris,[24] in reporting a series of 149 cases, noted complications varying from permanent sequela to death in 57 per cent of the cases. Complications arise because of the immediate cerebral ischemia in the territory supplied by the internal carotid artery or because of delayed embolism or thrombus from the site of ligation.

Rogers[25] reported that ligation of the internal carotid artery is to be avoided as a primary procedure. Dandy,[26] in evaluating the inefficiency of this procedure in controlling bleeding, classified it as a "preoperative procedure." This was due to the rapid re-establishment of cerebral vascular pressure following ligation and thus its inefficiency in controlling ethmoidal bleeding.

In the past 50 years ligation of the external carotid artery has been advocated and commonly accepted as the method of choice for control of severe nasal bleeding. This development is probably related more to the recognition that its terminal branch, the sphenopalatine artery, is the most frequent source of bleeding than to the evaluation of the physiological efficiency of the procedure; however, there remain a number of uncertainties related to the procedure, particularly with respect to the long-term prognosis and the effectiveness of such proximal ligation in controlling bleeding from a main artery with a rich collateral circulation.

Available data is particularly meager with respect to the long-term prognosis of this procedure. Barker,[27] Johnson and Foster[28] reported the recurrence of nasal bleeding due to re-establishment of collateral circulation within a week after ligation. These results seem comparable to the situation expressed by Dandy in evaluating the effectiveness of internal carotid artery ligation. The surgical axioms for control of hemorrhage established by surgeons of accepted reputations and experimental studies do not sustain this procedure.

Halsted,[29] in discussing surgical ligation, stated that the closer to the heart the ligature is placed, the greater is the likelihood that collateral circulation will be re-established. The physiologic response to such a ligation is a rise in blood pressure and a dilatation of the collateral circulation to compensate for the loss induced. The segment distal to the ligature

shows an immediate loss of pressure, followed by gradual rise and the return of pulsation.[30] While some authors[31-33] advocate this procedure because of its simplicity, difficulties and complications have been reported. Difficulty of exposure due to abnormally high division of the common carotid artery in the neck was reported by Davis,[34] who also noted frequent difficulty due to an overlying venous plexus.

Laryngeal and pharyngeal edema, unilateral paralysis of the tongue, and wound infection requiring tracheotomy have been reported as complications of the procedure.[7] Recurrence of bleeding has been reported frequently.[27-28] Some authors[35-36] claim that where bleeding is uncontrolled by external carotid ligation, a secondary procedure for ligation of the ethmoid artery through the orbital approach has been effective. This assumes mis-diagnosis of the bleeding point as the reason for failure.

Hirsch[37] and Davis,[34] however, admitting the existence of the ethmoid artery as a source of bleeding, explain the recurrence of bleeding as a result of the efficient collateral circulation. They, therefore, advocate the procedure of trans-antral ligation of the internal maxillary artery, as described by Seifert,[38] as a more effective procedure in that it affords an opportunity to cut off the arterial supply as close as possible to the site of hemorrhage, thus obviating the intermediate anastomotic branches.

Anterior ethmoid and internal maxillary artery ligation, as advocated by Hirsch,[37] conforms to the surgical principles of terminal ligation of vessels for control of hemorrhage. Criticism of the procedure, based upon technical difficulties, seems to be unwarranted by experienced rhinologists. Perhaps the words of one of our eminent surgeons are apropos: "It is frequently not the patient but the surgeon who is inoperable."[39]

The close relationship of the ethmo-sphenopalatine anastomosis and the severity of hemorrhage frequently preclude an exact diagnosis as to the origin of bleeding. Ligation of the external carotid or internal maxillary artery, at any point along its course is, therefore, commonly undertaken on the basis of frequency of bleeding from the sphenopalatine artery.

Where such a procedure fails a second operative approach for ethmoidal ligation becomes necessary.

As the result of my experience and that of others reviewed above, it became apparent that many surgical methods used to date for control of nasal hemorrhage have resulted in undesirable side effects and not infrequently have failed to achieve the primary aim of adequately controlling the hemorrhage. In order to circumvent these objections a modification of the fronto-ethmoid approach for exenteration of sinus disease, as originally advocated by Yankauer[40] and modified by Lynch, Ferris-Smith and others, was first employed by McCollough and myself in 1947.

Because of the psychologic stress and apprehension of the patient, we perform the operation under general anesthesia after temporary control of bleeding by means of nasal packing. The anterior ethmoid artery is exposed through a fronto-ethmoid incision. This exposure is easily maintained with the all-purpose, self-retaining retractor of McCollough. Non-absorbable material, silk or cotton, is preferable for ligating the artery. Placement of the ligature about the artery with the carrier and tier of Ferris-Smith simplifies the procedure. The efficiency of this ligature can be tested by removing all nasal packing and noting the effect on the bleeding when the ligature is tied.

If bleeding is arrested, the surgical procedure may be considered completed. If bleeding persists, the ethmoid labyrinth is exenterated, and the anterior wall of the sphenoid sinus is exposed. The mucosa is elevated from the anterior inferior wall of the sphenoid sinus, and a silk ligature inserted through the extreme lateral position so as to include the sphenopalatine artery as it enters the nose from its foramen. This ligature is also inserted with the ligature carrier and tier. All nasal packing is then removed to establish the fact that bleeding is controlled.

Since this procedure was adopted in 1947 it has been utilized in 12 cases of serious epistaxis when spot packing under anesthesia was ineffective. A survey of the cases (see Chart 1) operated by the fronto-ethmoid approach reveals that ligation of the anterior ethmoid artery or both the anterior ethmoid

CHART 1. SUMMARY—CASES OF SEVERE EPISTAXIS REQUIRING LIGATION

Case	Sex-Age	History	Therapy and Result
1. E. D. 1947	M. 58	Recurring left sided bleeding for two weeks. Uncontrolled with precision packing under anesthesia during 17 days of hospitalization.	Ligation of anterior ethmoid and sphenopalatine left. No packing. Discharged four days post-operative. No recurrence. Cured.
2. E. G. 1949	F. 45	Recurring bleeding for many years with three severe episodes requiring hospitalization. Unable to maintain her job and worried about recurrence. Always left sided.	Ligation left anterior ethmoid and sphenopalatine. No packing. No recurrence. Cured.
3. W. T. 1950	M. 26	Recurring bleeding for one month treated by family doctor. Emergency admission with anterior and posterior packing. Ligation performed because of recurrence of bleeding and repeated therapy with anxiety.	Ligation of left anterior ethmoid and sphenopalatine artery performed. No post-operative bleeding. Secondary finding at operation malignant nasopharyngeal tumor treated with X-ray. Living and well.
4. G. B. 1950	F. 60	Persistent bleeding, secondary to hypertension, in spite of packing. Bleeding point superior.	Anterior ethmoid ligated with control of bleeding. No post-operative packing. Discharged in five days. Cured.
5. R. M. 1951	M. 56	Persistent bleeding secondary to hypertension. Packing not effective. Patient apprehensive. Bleeding point posterior, right.	Anterior ethmoid and sphenopalatine ligation, right. Discharged five days post-operatively. Recurrence of mild bleeding 1953 and again in 1954. Controlled with packing. Hospitalized 72 hours and 96 hours.
6. B. M. 1951	M. 55	Recurring bleeding for 30 years, requiring frequent hospitalization and transfusions. Previous therapy included submucous, coagulation and packing. Twenty transfusions during one hospitalization. Eighteen transfusions at present hospitalization. Bleeding bilateral. Secondary to idiopathic telangiectasia.	Two-stage bilateral ligation nine days apart. Severe bleeding satisfactorily controlled. Discharged 18 days post-operatively. Has had mild episodes of bleeding from septal branches of external maxillary artery in anterior nasal space. Controlled $\bar{c}$ cautery and packing.

CHART 1. SUMMARY—CASES OF SEVERE EPISTAXIS REQUIRING LIGATION Continued

Case	Sex-Age	History	Therapy and Result
7. N. B. 1952	M. 38	Recurring bleeding for one week secondary to hypertension. Admission to the hospital after repeated packing and care by family physician. R.B.C. 2,500,000—H by 50%. Bleeding persisted in spite of precision packing. R.B.C. 1,890,000.	Ligation left anterior ethmoid and sphenopalatine. Discharged five days post-operative. No recurrence. Cured.
8. A. L. 1952	M. 58	Bleeding posteriorly. Hypertension, hyperplastic sinusitis and nasal polypi, right. Persistent bleeding in spite of precision packing.	Ligation right anterior ethmoid and sphenopalatine artery. Discharged five days post-operative. No recurrence. Cured.
9. T. G. 1952	M. 43	Recurring bleeding of undetermined origin uncontrolled by repeated nasal packing. Associated hyperplastic sinusitis and polypi.	Ligation right anterior ethmoid and sphenopalatine artery. No packing post-operative. Discharged six days after surgery. No recurrence. Cured.
10. F. H. 1953	M. 45	Repeated nasal bleeding for 24 hours. R.B.C. on admission 2,860,000. Persistent bleeding in spite of packing on repeated occasions.	Ligation right anterior ethmoid and sphenopalatine artery. Discharged seven days post-operative. No recurrence. Cured.
11. J. M. 1953	M. 46	Recurring bleeding over period of three weeks in spite of packing under anesthesia.	Ligation anterior ethmoid with complete control and discharge from the hospital four days post-operative. No recurrence. Cured.
12. A. M. 1954	M. 56	Recurring bleeding for 34 years because of familial telangiectasia. Hospitalized in Medical Service Veterans Hospital in February, 1954. Repeated nasal packing and recurrence of bleeding until anxiety of patient demanded more definitive treatment.	Ligation anterior ethmoid and sphenopalatine with complete post-operative control and release from the hospital one week post-operative. No recurrence.

and sphenopalatine arteries has been completely effective for control of hemorrhage in all cases to date. Post-operative period has been free of complication, and cardio-vascular and psychologic stresses have been neutralized.

One patient, aged 55, with a history of epistaxis for 30 years and two episodes of hospitalization for uncontrollable bleeding, requiring as high as 20 blood transfusions, had bilateral ligation performed within a period of one week. In the three years following surgical ligation he has been free of bleeding. Another case of severe nasal bleeding secondary to congenital telangiectasia had required repeated transfusions in the course of 15 hospital admissions during the past seven years. There has been no further bleeding since this procedure was performed eight months ago.

SUMMARY.

Serious epistaxis has been presented as a problem worthy of evaluation because of the frequency of its occurrence, distress to the patient, questionable therapy, and serious sequelae. An attempt has been made to elucidate the adequacies and inadequacies of past surgical procedures from the standpoint of acceptable surgical axioms for the permanent control of hemorrhage.

The use of the fronto-ethmoid approach as a one-stage procedure for terminal ligation of the anterior ethmoid and sphenopalatine arteries is offered as an alternative to the two-stage procedure of ethmoid artery ligation when trans-antral ligation of the internal maxillary artery is ineffective.

The justification for use of the fronto-ethmoid approach is based upon the clinical results of the 12 cases reported and the resolution of the anatomic and physiologic objections to the other surgical procedures.

The surgical procedure should be familiar to every rhinologist trained in the care of nasal and sinus disease. It should be part of the armamentarium of the rhinologist in controlling severe epistaxis when emergency packing and spot packing under anesthesia have failed.

CONCLUSION.

From this analysis of the past and present surgical care of severe nasal hemorrhage, the following conclusions are apparent:

1. Persistent and uncontrolled epistaxis may cause serious complications due to cardio-vascular stress.

2. Fatalities have been reported due to inadequate therapy.

3. During episodes of severe epistaxis, it is frequently impossible to determine the source of bleeding.

4. Persistent nasal bleeding creates a severe psychologic stress for the patient, making it desirable to utilize general anesthesia for carrying out therapy.

5. Severe nasal hemorrhage is usually associated with rupture of the anterior ethmoid or sphenopalatine arteries.

6. Permanent surgical control of nasal bleeding can be obtained by terminal ligation of vessels.

7. Ligation of a major vessel to control bleeding from its lesser branches is unsound.

8. Ligation of the common or internal carotid artery is inefficient and hazardous.

9. Ligation of the external carotid artery is not always a simple procedure and has been followed by undesirable complications.

10. Recurrence of nasal bleeding from collateral circulation subsequent to external carotid ligation has been reported.

11. Secondary procedures for ethmoid artery ligation have frequently been necessary due to "mis-diagnosis" of the bleeding point.

12. Terminal ligation of vessels for effective control of nasal hemorrhage can be accomplished through the fronto-ethmoid approach.

13. Criticism of a rhinologic surgical procedure on the basis of technical difficulties is unwarranted by those adequately trained in rhinology.

BIBLIOGRAPHY.

1. Houser, K. M.: Oxidized Cellulose Gauze Packing. *Jour. A.M.A.*, 132:143, Sept. 21, 1946.

2. Fox, S. .: Treatment of Epistaxis by Sclerosing Injections. The Laryngoscope, 54:398, Aug., 1944.

3. Kler, J. H.: Use of Absorbable Gauze. *Arch. Otolaryngol.*, 46:801, Dec., 1947.

4. Cope, B. A., and Hipskind, M. M.: Use of New Agents in Treatment of Epistaxis. *Eye, Ear, Nose and Throat Monthly*, 26:417, Aug., 1947.

5. Dixon, O. J.: Epistaxis—Use of Stent Marine Sponge. *Jour. Missouri M.A.*, 45:824, Nov., 1948.

6. Ogura, J. H., and Senturia, B. H.: Epistaxis. The Laryngoscope, 59:743, July, 1949.

7. Hallberg, O. E.: Severe Nosebleed and Its Treatment. *Jour. A.M.A.*, 48:355, Feb. 2, 1952.

8. Weddell, B.; Macbeth, R. G.; Sharp, H. S., and Calvert, C. A.: Surgical Treatment of Severe Epistaxis. *Brit. Jour. Surg.*, 33:387, April, 1946.

9. McArthur, J. E.: Recurrent Massive Epistaxis. *Canadian Med. Assoc. Jour.*, 57:384, Oct., 1947.

10. Abraham, B. H.: Ligation of External Carotid Artery for Persistent Nasal Hemorrhage. *Arch. Otolaryngol.*, 8:29, July, 1928.

11. Shenkin, H. A.; Cabreseis, F.; Vander Nordt, G.; Sayers, C., and Cooperman, R.: Hemodynamic Effect of Unilateral Carotid Ligation on the Cerebral Circulation of Man. *Jour. Neurosurgery*, 8:38, Jan., 1951.

12. Dandy, W. E.: Results Following Bands and Ligatures on Human Internal Carotid Artery. *Ann. Surg.*, 123:384, March, 1946.

13. Sweet, W. H., and Bennett, H. S.: Changes in Carotid Pressure with Occlusion and Their Clinical Significance. *Jour. Neurosurg.*, 5:178, March, 1948.

14. Matson, D. D., and Woodhall, B.: Intracranial and Cervical Trap Ligation of the Carotid Artery. *Jour. Neurosurg.*, 8:567, 1948.

15. Walsh, F. B., and King, A. B.: Ocular Signs of Intracranial Saccular Aneurysm—Experimental Work of Collateral Circulation through the Ophthalmic Artery. *Arch. Ophthalmol.*, 27:1, Jan., 1942.

16. Gray, H.: Anatomy of the Human Body.

17. Sugarbaker, E. D., and Wiley, H. M.: Simultaneous Tourniquet or Temporary Ligation of Both External Carotid Arteries. *Surg.*, 29:296, Feb., 1951.

18. Chambers, W. R.: Value of External Carotid Angiography in Surgery of Brain Tumor. *Jour. Indiana M.A.*, 44:23, June, 1951.

19. Babcock, W. W.: Textbook of Surgery, p. 78.

20. Abernathy: Quoted by Spar-Hollberg. Severe Epistaxis and Its Management. *Ann. Otol., Rhinol., and Laryngol.*, 56:141, 1947.

21. KERWIN, R. W.: Carotid Artery Surgery. *Arch. Otolaryngol.*, 35:30, Jan., 1942.

22. WOODRUFF, G. H.: Massive Pharyngeal Hemorrhage—Ligation of Carotid Arteries. *Ann. Otol., Rhinol., and Laryngol.*, 54:202, March, 1945.

23. SARGENT, F. B.: Recurring Severe Hemorrhage from Nose and Pharynx. *Ann. Otol., Rhinol., and Laryngol.*, 54:495, Sept., 1945.

24. VORIS, HAROLD C.: Complications of Ligation of the Internal Carotid Artery. *Jour. Neurosurg.*, 8:119, 1951.

25. ROGERS, LAMBERT: Ligation of the Common Carotid Artery. *Lancet*, 1:949, June 4, 1949.

26. DANDY, W. E: Results Following Ligation of the Internal Carotid Artery. *Arch. Surg.*, 45:521, 1942.

27. BARKER, G. N.: Serious Epistaxis. *Jour. Laryngol. and Otol.*, 58:293, July, 1943.

28. JOHNSON, M. C., and FOSTER, M. E.: Ligation of the External Carotid Artery for Traumatic Nasal Hemorrhage. *Ann. Otol., Rhinol., and Laryngol.*, 42:588, June, 1933.

29. HALSTED, W. S.: *Jour. Exper. Med.*, 24:171, 1916.

30. MCKETTRICK, O. F., and BARTLETT, W.: A Study of Secondary Hemorrhage Treated by Ligation of the Common Carotid Artery. *Ann. Surg.*, 65:715, 1917.

31. MACBETH, R. G.: Anterior Ethmoid Hemorrhage. *Jour. Laryngol. and Otol.*, 62:242, Jan., 1948.

32. MCKNIGHT, H.: Carotid Ligation in Epistaxis. *Texas State Jour. Med.*, 22:271, Aug., 1926.

33. HYDE, F. T.: Ligation of the External Carotid Artery for Control of Idiopathic Nasal Hemorrhage. THE LARYNGOSCOPE, 35:899, Dec., 1925.

34. DAVIS, E. D.D.: Ligature of the Internal Maxillary Artery through the Antrum for Uncontrollable Epistaxis. *Jour. Laryngol. and Otol.*, 60: 420, 1945.

35. PELUSE, S., and FISHLER, H. W.: Epistaxis Controlled by Combined Ligation of the External Carotid and Anterior Ethmoidal Arteries. *Arch. Otolaryngol.*, 60:70, July, 1954.

36. WILLIAMS, J. L.: Ethmoidal Epistaxis Necessitating Operation. *Jour. Laryngol. and Otol.*, 60:292, July, 1945.

37. HIRSCH, C.: Ligation of Internal Maxillary Artery in Patients with Nasal Hemorrhage. *Arch. Otolaryngol.*, 24:589, Nov., 1936.

38. SEIFFERT, ALFRED: Unterbindung der Arteria Maxillaris Interna. *Ztschr. F. Hals, Nasen-u. Ohrenh.*, 22:323, 1929.

39. MARTIN, H.: Instruction Course, *Amer. Acad. Ophthal., and Otolaryngol.*, 1952.

40. YANKAUER: Complete Spheno-ethmoid Operation. THE LARYNGOSCOPE, 31:831, 1921.

CANCER OF THE EXTERNAL AUDITORY MEATUS.*†

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Cancer of the ear, nose or throat is a morbid disease which is less or more morbid, depending on several specific factors. The differentiation of the cells, the rate of growth, the rapidity of metastasis, and the bio-physical bed of the host, all decrease or increase the chances for cure; however, one factor concerning cancer of the auricle, so important that it is the moving force for the investigation reported in this paper, is that of location.

Innumerable articles have been written about the poor prognoses of cancer located in the posterior third of the larynx as compared with the anterior third, even when exactly the same pathology may be observed under the microscope. This same factor, location, seems to be of even greater importance in cancer of the auricle which develops within 1 centimeter of the external auditory meatus. This must be pointed out because the degree of radicalism employed by the otologic surgeons depends upon the possible extent of the tumor he visualizes grossly.

The most common types of cancer in this area are the squamous cell carcinoma, the basal cell carcinoma, and the cylindroma.

In our series of 88 cases we have 45 squamous cell carcinomas and 35 basal cell carcinomas, which together make up 91 per cent of the entire series. There were also two cylindromas and two adenocarcinomas. It is not pertinent to this paper to discuss other less common types of cancer which have been found in this area.

Although it is well known that the epidermoid carcinoma is much more likely to metastasize by way of the lymphatics into

* Read at the Fifty-Ninth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Hollywood, Fla., March 17, 1955.

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Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication March 23, 1955.

the neck, many of us have been lulled into a false sense of relative security by the pathologist's report of the so-called low grade malignancy called basal cell carcinoma. It is an established fact that such a lesion in other areas of the head, including the periphery of the auricle, is usually a low grade, slowly growing affair that can easily be handled in most instances either by X-radiation or with a 1 centimeter margin if treated surgically; however, our own experience teaches us that basal cell carcinoma within 1 centimeter of the external auditory meatus may act either like a docile lamb, a fox, or occasionally an octopus.

Basal cell carcinoma may remain localized, and even after being erroneously treated with ointments and antibiotic preparations by an unknowing physician over a long period of time, eventually can be removed safely with a 1 centimeter margin, and never recur. This can be called the docile lamb type.

On the other hand a basal cell carcinoma with the same gross appearance and showing an ulcerated center, unknown to the physician, may surreptitiously grow beneath the surrounding skin for a distance of 2 to 3 centimeters and not even be palpable to the most careful observer. This may be called the fox-like type.

Finally, the type that gives the most concern is the basal cell carcinoma that may also present the same localized small ulceration described previously, but like an octopus, infiltrates and sends its cells along the peri-neural lymphatic pathways, dichotomous-like, and become the most difficult to eradicate.

At this point I must confess that I have not been able to determine clinically early enough what might be the nature of the beast. If this were possible then some of the conclusions about the proper therapy, drawn from my observations, would no longer be valid.

Figure 1 tells a part of the sad story of a man who was subjected to repeated surgery for basal cell carcinoma over a period of 17 years and finally culminated in death, due to extension of disease to the cerebellum. The disease process at the last procedure performed revealed the cancer cells to have

penetrated to the vertex of the scalp, 6 centimeters from the ulcer periphery, with no clinical evidence of such disease being present at this point.

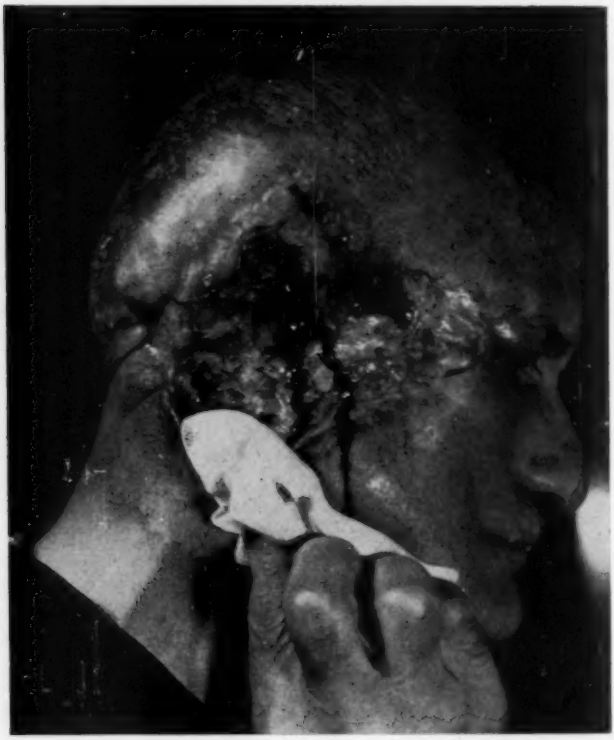


Fig. 1.

A word about the cylindroma. The pathologist as well as the clinician must be extremely careful to distinguish between the so-called basal cell carcinoma of skin origin and the cylindroma that finds its way via the anterior portion of the external auditory meatus from its site of origin in the parotid gland. Not infrequently there is no clinical evidence for swelling of the parotid gland in the region of the tragus, and

yet an ulcerated tumor mass may present itself within 1 centimeter of the external meatus.

One of our cases was treated by local excision at first for what was reported as basal cell carcinoma of the external meatus, but as recurrence took place within a few months it became obvious that the tumor was a cylindroma that arose in the tail of the parotid gland and worked its way into the external auditory meatus. More radical surgery was, therefore, undertaken.

It is self evident in such cases that surgery of the auricle must also include excision of the parotid gland, even sacrificing the facial nerve if essential for establishing a cure. The first operation is usually the one real chance for cure in these cases.

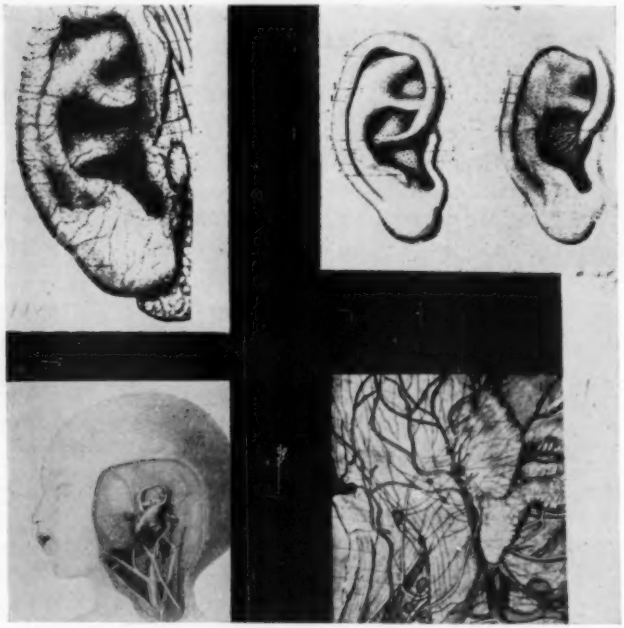
The high rate of failure to produce cures in cancer of the auricle in which the disease appears within a centimeter of the external auditory meatus compares unfavorably with statistics for cures in other areas within the realm of otolaryngology. The cure rate definitely is not reflected in the 95 per cent cures obtained in skin cancer in general.

The material for this report was drawn from the records of 88 cases treated over the past 25 years at the Massachusetts Eye and Ear Infirmary, the Massachusetts General Hospital, and Pondville Hospital, a Massachusetts state institution devoted entirely to cancer work. All cases which did not specifically present themselves as neoplasm within 1 centimeter of the external auditory meatus were excluded on the clinical basis that lesions of the periphery of the auricle usually respond to therapy as well as skin cancer in general and, therefore, were not pertinent to this report.

The location of the tumor near the external meatus is more serious, partly because of the nature of the lymphatics in this region. For example, Fig. 2 A and D represents Belov's demonstration of the lymphatics of the auricle and external meatus. This cutaneous network contains three groups: the first, a posterior group, included the region of the helix, the antihelix, triangular fossa, the whole of the internal surface and the posterior portion of the external auditory meatus. They

number 6 to 12 and end in the mastoid glands for the most part; some by-pass them and go directly to the upper deep cervical nodes.

(A) Belov. (B) Sappey's.



(C) Most. Fig. 2. (D) Belov.

(A and D) BELOV: Altas de Anatomie del organa del oida y de L. & S. Region con e vinculadas. Buenos Aires, Oceana, 1930.

(B) SAPPEY: Traite d'anatomie descriptive, Ed. Vol. 3, Paris, Lecrosnier et Babe, (Fig. 727), 1889.

(C) MOST, AUGUST: Die Topographie de Lymphgefass und Apparate des Kopfes und des Halses. Berlin, 1906.

The second, the inferior group, drains the inferior portion of the external auditory meatus, the lobule, and adjacent portions of the auricle. Some drain into the sub parotid group and some directly to the upper deep cervicals.

The third and anterior group contains four to six trunks and drain the concha and anterior wall of the external meatus and empty into the pre-auricular glands.

Figure 2 B represents a beautiful dye injection demonstration by Sappey, and shows the tremendous number of lymphatics within a 1 centimeter area of the external auditory meatus. With this picture in mind it is not difficult to presuppose that cancer in this region can readily be a more morbid disease.

This photograph demonstrates the mastoid or retro-auricular glands, usually two in number, that lie on the mastoid insertion of the sternomastoid muscle. These drain the temporal portion of the scalp, the internal surface of the auricle with the exception of the lobule, and also receive afferents from the posterior surface of the external auditory meatus. The efferents from this gland go to the upper deep cervical group under the sternomastoid muscle.

In determining what happened to these 88 patients it must be pointed out that not only otologists but also general surgeons treated many of them. Forty-one of the cases were treated by general surgeons and 26 were treated by otologic surgeons. The rest were treated with X-radiation. The oldest patient in this group at the age of onset of the disease was 94, the youngest 32. A further breakdown in this age group, pertinent in view of the numbers in the middle age group, is shown in Table I.

TABLE I — AGE OF ONSET.

30-39 years old	5
40-49 years old	12
50-59 years old	13
60-69 years old	31
Over 70 years of age	27

About one third of the cases occurred in those under the age of 60. Incidentally 65 were males compared with 23 females, a ratio of about 3:1.

TABLE II — SURVIVAL RATE.

No. of patients in series	88
No. of survivals without clinical evidence of cancer:	
5 years	35
7 years	22
10 years	14

TOTAL—15.8 PER CENT 10-YEAR CURE RATE.

The usual criteria for five-year survival without obvious recurrence did not seem to apply to cancer in this area. This is significant when one reports the so-called five-year survivals and notes the dwindling figures as seven years and ten years are reached; however, this chart deserves further clarification. Many of the 74 uncured patients continued to live with disease for as long as 15 or more years, receiving intermittent treatment by surgery or radiation at one time or another; also, two-thirds were 60 years of age or older, and died of other disease despite the persistence of the ear cancer.

The criteria as to the size of the presenting lesion were reviewed, and it is of striking interest that size mattered very little, since the largest and smallest groups showed the same poor end results. Let us break this down further.

TABLE III — SIZE OF PRESENTING LESION.

Size — less than 1.0 cm.	11 in series
5 year survival without obvious disease	4
7 year survival without obvious disease	2
10 year survival without obvious disease	1
Size — 1.0 cm. to 2.0 cm.	39 in series
5 year survival without obvious disease	20
7 year survival without obvious disease	13
10 year survival without obvious disease	9
Size — 2.5 cm. to 5.0 cm.	25 in series
5 year survival without obvious disease	9
7 year survival without obvious disease	6
10 year survival without obvious disease	3
Size — larger than 5.0 cm.	9 in series
5 year survival without obvious disease	2
7 year survival without obvious disease	1
10 year survival without obvious disease	1

Even though most of the 10 year survivals had 1.0 to 5.0 centimeter size lesions at the onset, the poorer showing of the smallest and largest lesions demonstrates that very little importance can be attached to the relative size of the presenting lesion in this area.

What about the pathology insofar as chances for cure are concerned?

TABLE IV — PATHOLOGY.

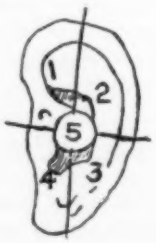
Basal cell	35 cases
5 year survival without obvious disease	21
7 year survival without obvious disease	15
10 year survival without obvious disease	9
Squamous cell	45 cases
5 year survival without obvious disease	12
7 year survival without obvious disease	6
10 year survival without obvious disease	4
Cylindroma	2 cases
5 year survival without obvious disease	1
Adenocarcinoma	2 cases
10 year survival without obvious disease	1

The type of pathology in relation to cure shows a ratio of about 2.8 basal cell carcinoma 10-year cures to one of squamous cell origin. One would ordinarily expect a higher ratio of basal cell cures, in view of the relative benignity of this neoplasm in other areas of the skin.

In view of the lymphatic distribution it seems worth while to break down the quadrants of the region of the external auditory meatus and note the cures per quadrant in order to appreciate better the significance of quadrant location of the tumor area involved.

TABLE V — QUADRANT LOCATION.

Quadrant	No. pts.	5 years	7 years	10 years
1	1	0	0	0
3	4	2	1	0
4	2	1	0	0
5	28	12	8	6
1,2	5	1	0	0
1,4	14	8	6	4
2,3	13	5	4	2
3,4	11	5	3	2
1,4,5	1	0	0	0
3,4,5	1	0	0	0
1,2,3,4	4	1	0	0



This demonstrates diagramatically the quadrant distribution used and the gross quadrant occupancy of the lesions. It is evident that not enough cases of tumor in any one quadrant survived to suggest that quadrant location is of any great significance.

Could it be possible that more cures were obtained in this series when the surgery was done by the otologist rather than the general surgeon? One would expect a difference in the end-results on the basis of the special training of the otologist in this area. Our statistics did not bear out my clinical impression that there might be a great difference; however, these statistics do not tell the whole story. Some of these cases had had previous local surgery or radiation, and many had had prolonged medical local treatment prior to reaching the otologic surgeon. In some cases there was a lapse of two years which may account for some of the poor showing by the otologists. Many skilled otologic and general surgeons are not so well oriented in cancer problems as those who have special interests in this field. Many of these 88 cases were treated by surgeons who lacked special orientation in cancer problems. The phrase "en bloc" in cancer surgery demands the most meticulous surgical technique in spite of the anatomical difficulties encountered.

Table VI demonstrates the survival rates in the patients treated by the general surgeon as well as by the otologist.

TABLE VI — SURVIVAL RATE — GENERAL SURGEON AND OTOLOGIST

General surgeon	41	cases
5 year survival without obvious disease	21	
7 year survival without obvious disease	13	
10 year survival without obvious disease	6	
Otologist	26	cases
5 year survival without obvious disease	8	
7 year survival without obvious disease	4	
10 year survival without obvious disease	3	

Has the type of surgery performed affected the end-results? It is my personal conviction that when cancer is present in the region of the external auditory meatus it should be

removed *en bloc,* including the underlying periosteum and mastoid cortex, along with most of the auricle. A modified radical mastoidectomy, including most of the auricle and as much bone tissue *en bloc* as possible, would seem to offer the patient the best possible chance for cure. More of this type of surgery has been done within the past five years. The efficacy of this approach is not proven in this next table, mainly because of the lack of a sufficient number of cases so treated over a long enough period of time. In view of the high rate of failures more radical *en bloc* surgery becomes imperative.

TABLE VII — TYPE OF TREATMENT.

Mastoid operation	18*
5 year survival without obvious disease	2
7 year survival without obvious disease	1
10 year survival without obvious disease	0
Local Excision	49
5 year survival without obvious disease	27
7 year survival without obvious disease	16
10 year survival without obvious disease	9
Treated by X-ray or radium	21
10 year survival without obvious disease	5

* Six of these have been done within the last 5 years, so cannot be included in 5 year survival; one died of unrelated disease.

The poor results obtained by surgery including mastoidectomy in past years must be interpreted in the light of the following chart (see Table VIII).

Only four of these 18 cases had had prior attempt at removal of the lesion by surgery or radiation; in many patients the disease had gone unrecognized for several years. Many came to the otologist long after the onset of the disease because of the failure of early diagnosis. Most of the mastoid surgery undertaken then was not *en bloc,* and although this type of procedure was adequate for infection, it was not sufficiently effective for the treatment of cancer.

The high rate of failures leads me to suspect that more radical surgery during the first procedure might reduce that figure immeasurably. It has always been felt that the first operation gives the patient the best chance for cure. The need for secondary operating because of recurrence has usually

meant a much poorer prognosis. This fact was borne out in the survivors in this series. Very few survivors had more than one operation.

TABLE VIII — THE 18 MASTOID CASES.

	Previous Treatment	Survival after Operation	Pathology	Location	Size
1.	Treated for otitis (4 yrs.)	8 months	Cylindroma	5	2.0
2.	X-ray and cautery (4 yrs.)	5½ years	Basal Cell	1,4	4.0
3.	2 local excisions	1 yr., 7 months	Ep. Ca. III	5	4.0
4.	Treated for furunculosis (1 yr.)	1 yr., 1 month	Ep. Ca. I	5	1.0
5.*	Treated for eczema ("for years")	2½ yrs. (living without disease)	Ep. Ca. II	5	2.0
6.*	None	3 yrs., 2 months (living without disease)	Ep. Ca. I	5	0.4
7.	Biopsy only	6 months (died ca. stomach)	Ep. Ca. III	2,3	0.5
8.*	Biopsy only	3 yrs., 1 month (living without disease)	Ep. Ca. I	5	1.0
9.*	None	2½ yrs. (living without disease)	Ep. Ca. I	5	0.6
10.	None	1 yr., 4 months	Adenocarcinoma	5	2.0
11.*	Penicillin only	2 yrs., 2 months (living without disease)	Ep. Ca. III	2,3	4.0
12.	Treated for eczema	1½ yrs.	Ep. Ca. III	5	0.5
13.	"drops"	1 yr., 1 month	Ep. Ca. I	5	1.0
14.*	None	1 yr., 3 months (living without disease)	Cylindroma	1,2,3,4	3.0
15.	"treated by L M D" (nature not stated)	7 yrs., 4 months (living without disease)	Basal Cell	5	1.0
16.	Biopsy only	1 yr., 4 months	Basal Cell	5	2.0
17.	2 excisions	3 yrs., 10 months	Basal Cell	5	2.0
18.	2 excisions and X-ray	6 yrs.	Basal Cell	1,4	2.0

* Treated within past 5 years.

Note: All patients (except those designated as "living without disease" died with disease in time specified under "Survival after Operation." The only exception is No. 7, who died of unrelated cancer of stomach six months after mastoidectomy.

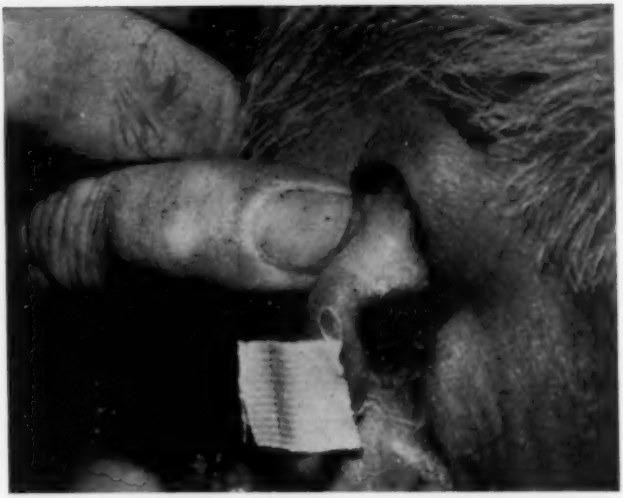
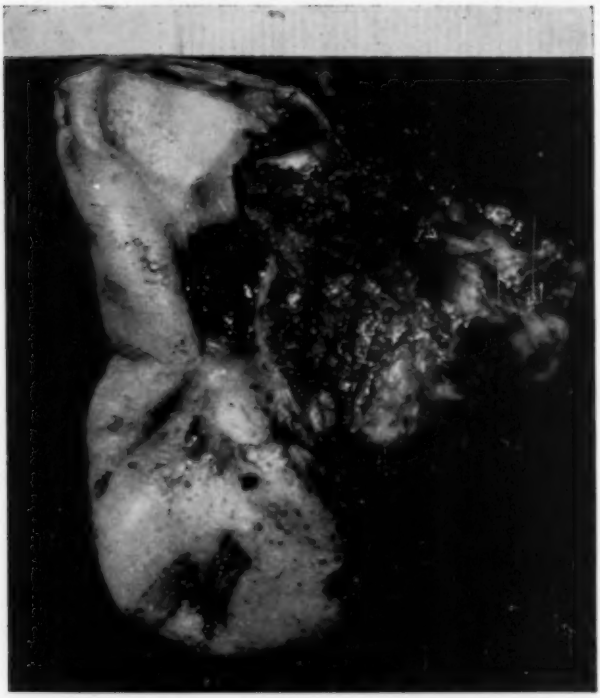


Fig. 3.

Superior.



Inferior. Fig. 4.

X-ray therapy shows a much higher number of survivors than might have been expected; however, X-radiation in an area of bone and cartilage is fraught with danger because of the possibility of radiation necrosis with its attending intense suffering, and it is often difficult to determine clinically, or by X-ray study, whether periosteum, perichondrium, cartilage or bone is involved. Since the actual extent of the disease cannot

Fig. 5.

always be determined, wide *en bloc* surgery, including most of the mastoid process and most of the auricle, offers the best chance for complete eradication of the disease.

An example of the type of lesion with which our recommended therapy is concerned is shown in Figs. 3 and 4.

This patient, E. M., with a basal cell carcinoma within 1 centimeter of the external auditory meatus, had been treated prior to surgery unsuccessfully with two separate courses of X-ray therapy. Our surgery included most of the auricle and a resection of the mastoid cortex including skin and periosteum

of the external auditory canal *en bloc*, followed by piecemeal removal of most of the mastoid and then skin grafts. His prosthesis on patient in Fig. 5, made by Dr. Henry Carney, an oral surgeon of Boston, has been most satisfactory to the patient. It is made of pliable plastic material and can be worn in a shower and handled fairly vigorously without disturbing its status quo.

CONCLUSION.

Because of the many factors involved in this statistical study and the number of variants which could influence the significance of these factors, the conclusions I have reached may not be authentically established statistically; however, it can be stated that cancer of the auricle located within 1 centimeter of the external auditory meatus is a serious disease in spite of its histologic description, size, or quadrant location. It should be treated by radical surgery which should include removal of most of the auricle and the underlying mastoid cortex *en bloc*, and modified radical mastoidectomy in chosen cases.

Although it is true that some may be cured with X-radiation or local excision it is usually not possible to determine the true extent of the disease. For this reason the best possible procedure that can be performed as the first operation should be the treatment of choice. This may seem radical esthetically because of the removal of most of the auricle, but it is not at all radical when one considers the high rate of failures. This more extensive surgery is essential to increase the present poor 10 year survival rate of 15.8 per cent to a level consistent with results obtained from treatment of cancer of the skin elsewhere on the body.

HEADACHE, SINUSITIS, AND MOTHER GOOSE.*†

JAMES T. KING, M.D.,

Atlanta, Ga.

" . . . And when the child was grown, it fell on a day, that he went out to his father to the reapers. And he said unto his father, My head, my head. And he said to his servant, carry him to his mother. And when he had taken him, and brought him to his mother, he sat on her knees till noon, and then died . . . " (II KINGS *4:18-20)*

Anyone making a talk on how to cure headache by treating the nasal accessory sinuses could well take his text from II Kings, fourth chapter, 19th verse, using these four words—"My head, my head." This passage would apply not only to the patient but to the physician treating the patient as well.

In my experience with headache and sinusitis I have been impressed, not by how many headaches are caused by sinus disease, but how few. In the other branches of medicine, and among the public as well, sinus disease has always been regarded as a common cause of headache. In my locale, this impression still persists. As we all know, the causes of headache are almost innumerable; sinusitis is only one of them. To illustrate this point I have taken from my files the records of 460 consecutive patients (1948-1951), whose chief complaint was headache and in whom the presence of sinusitis was either suspected or assumed. I have analyzed and grouped these patients according to the cause of their symptoms.

1. Nothing Group—173 cases, or 38 per cent. This, my largest class, composes what I have chosen to call the "nothing" group. In these cases there was nothing about the history or clinical examination that permitted the diagnosis of any disease or syndrome. Often there was nothing more apparent

* Read at the meeting of the Southern Section, American Laryngological, Rhinological and Otological Society, Inc., Charlottesville, Va., January 22, 1955.

† From the Department of Otolaryngology, Emory University School of Medicine.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, February 23, 1955.

than that the patient was having financial difficulties. Sometimes husband and wife were fighting; others merely had hangovers.

2. *Neuralgia Group*—123 cases, or 27 per cent. In the second largest group are those cases with neuralgia of one or more divisions of the Vth Cranial Nerve. This group did not differ greatly from the first group, as the same strong psychosomatic factor was present; however, these patients did present some reason for the suspicion of sinusitis. When the frontal nerve was involved, the pain and tenderness of frontal sinusitis was to a certain extent simulated, and when the maxillary nerve was involved, the pain and tenderness of maxillary sinusitis was partially imitated.

3. *General Systemic Group*—80 cases, or 17 per cent. These patients were shown to have such things as allergy, colds, influenza, or hypertension as a cause of their headache.

4. *Vascular Group*—33 cases, or 7 per cent. Twenty-two cases of migraine and 11 of histamine cephalgia comprised this group. In compiling these figures, I was somewhat surprised to find that these two disorders contributed such a small percentage to the total number.

5. *Intracranial Disease Group*—19 cases, or 4 per cent. These included head injury in 11 cases, cerebral hemorrhage in three cases, brain tumor in two cases and meningitis in two cases. The nineteenth patient turned out to have multiple sclerosis as a presumable cause of her headache.

6. *Sinusitis Group*—32 cases, or 7 per cent. You will note that, contrary to common belief, sinusitis provided the second smallest group—only the intracranial diseases provided fewer headaches. Out of every 14 patients in whom the headache was thought to come from the sinuses, only one was actually proven to have sinusitis.

These six groups compose the entire 460 cases; however, this classification could easily be changed to include yet another group. This group would be composed of those patients with chronic headache, who go from one doctor to another receiving various treatments, but never a cure, and who have been examined repeatedly and no real pathology has been

found. In treating these patients in the early part of this series, my headache was as severe as theirs, since I failed to give them any permanent relief.

Sometimes, when I was enthusiastic about a certain treatment, the patient seemed benefited—possibly by the treatment, more probably by my enthusiasm. As enthusiasm waned, so did the beneficial effect. I also noted than on several occasions when I gave the patient a drug he had never received before, he would be helped by it initially. Almost always the headache would return, and when it did this particular drug was no longer helpful. Finally, there were those patients whom I thought that I had cured, since they did not return for further treatment. My therapeutic superiority complex was converted to an inferior status when I learned that they had only left me for consultation with one of my colleagues. Small comfort it was to learn subsequently that they had also left my colleague for someone else.

I have come to the conclusion that we should do something to change the attitude of these patients toward their trouble. Rather than give them any more histamine, ergot derivatives, vitamins, diathermy, argyrol packs, bloody nasal aspirations, or Epsom salt, I now merely try to impart a bit of the simplest philosophy I know, that of Mother Goose:

For every malady under the sun
There is a cure or there is none.
If there be one try to find it.
If there be none never mind it.

If we can get these particular patients to heed this rhyme it will help them more than the specific therapy presently available, unless we have the divine healing power of the prophet Elisha, who restored to life the child who died after crying, "My head, my head."

384 Peachtree Street, N. E.

UNIVERSITY OF ILLINOIS ANNUAL ASSEMBLY IN OTOLARYNGOLOGY.

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly of Otolaryngology from September 19 through October 1, 1955. This Assembly will consist of two parts:

Part I — September 19 through September 24, 1955, will be devoted to surgical anatomy of the head and neck, fundamental principles of neck surgery and histopathology of the ear, nose and throat. This week will be under the personal direction of Maurice F. Snitman, M.D.

Part II — September 26 through October 1, 1955, will be devoted entirely to lectures and panel discussion of advancements in otolaryngology. The chairman of this section will be Emaneul M. Skolnik, M.D.

Registration is optional for one or both weeks. For further information, address Dr. Francis L. Lederer, 1853 West Polk St., Chicago 13, Ill.

POST-GRADUATE COURSES AT TEMPLE UNIVERSITY.

The following Post-Graduate Courses to be given in this Department during the current year:

Post-Graduate Course in Broncho-Esophagology, June 13-24, 1955; October 17 - 28, 1955.

Post-Graduate Course in Laryngology and Laryngeal Surgery, September 19 - 30, 1955.

These courses are all to be given in the Department of Laryngology and Broncho-esophagology, Temple University Hospital and School of Medicine, under the direction of Dr. Chevalier L. Jackson and Dr. Charles M. Norris. The tuition fee for each course is $250.00. Further information and application blanks can be obtained from Dr. Chevalier L. Jackson, 3401 N. Broad Street, Philadelphia 40, Pennsylvania.

SOUTH AFRICAN LOGOPEDIC SOCIETY.

The South African Logopedic Society (Society of Speech Therapists) publishes a Journal in May and October of each year. Articles deal with the organic and psychological aspects of speech defects, and include subject matter of interest to the medical and allied professions.

Relevant contributions in the field of otolaryngology are invited.

Subscriptions are 10/– per annum including postage and should be addressed to: The Editor, Journal of the S. A. Logopedic Society, Witwatersrand University, Milner Park, Johannesburg, So. Africa.

COLBY COLLEGE — AUDIOLOGY FOR INDUSTRY.

Colby College, Waterville, Maine, presents the Third Annual Course in Industrial Deafness, August 7 - 13 inclusive. Objective of the course will be to train personnel in initiating and in conducting conservation hearing programs in noisy industries. Seven full time instructors have been selected from authorities in this field. Class limited to 20 participants.

Registrants will live on the College Campus and the Tuition fee of $200.00 includes board and room. Applications should be made to Mr. William A. Macomber, Director, Division of Adult Education and Extension, Colby College, Waterville, Maine. Frederick Thayer Hill, M.D., Director; Joseph Sataloff, M.D., Assistant Director.

BRONCHOESOPHAGOLOGY COURSE

The next Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for the period October 24 to November 5, 1955, under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

ACADEMY-INTERNATIONAL OF MEDICINE.

A completely revised Fourth Edition of "Professional Films" is now in compilation. (The frequency and number of future insert pages necessary to assure a comprehensive index that is continuously current over a period of years will be determined by the volume of forthcoming productions.) It will include new sections providing biographical data on authors, and information on the audio-visual activities of medical schools, dental schools and post-graduate teaching centers.

Over 28,000 copies of previous Editions are in use by medical and dental schools, Program Chairman of State and specialty societies, and others here and abroad. AIM provides this valuable audio-visual information to the profession-at-large, without profit, as one of its contributions toward elevating the standards of medical and dental services by expediting the dissemination of professional knowledge.

You are urged to assist by *1.* Informing film authors of this announcement so that they can write for questionnaires. *2.* Providing the film title and full name and address of any film author. Write to the Academy-International of Medicine, 601 Louisiana Street, Lawrence, Kansas.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Plans have been completed for a joint meeting of the North Carolina Eye, Ear, Nose and Throat Society and the South Carolina Society of Ophthalmology and Otolaryngology at Columbia, S. C., on September 12, 13, and 14, 1955. Headquarters will be the Columbia Hotel.

The following ophthalmologists will be on the program: Dr. E. W. D. Norton of New York, Dr. Frank Carroll of New York, and Dr. William B. Clark of New Orleans.

A most attractive program has been arranged. For further information address Roderick Macdonald, M.D., Secretary and Treasurer, 330 East Main Street, Rock Hill, S. C.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Wm. J. McNally, 1509 Sherbrooke St., West Montreal 25, Canada.
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Secretary-Treasurer: Dr. Lawrence R. Boies, 90 S. Ninth St., Minneapolis 2, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Seigniory Club, Montreal, Canada, May 11-12, 1956.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

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Librarian, Historian and Editor: Edwin N. Broyles, 1100 North Charles St., Baltimore, Md.
Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Dean M. Lierle, Iowa City, Iowa.
President-Elect: Dr. Percy Ireland, Toronto, Canada.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

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Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October 9-15, 1955.

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Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Sheraton Mount Royal Hotel, Montreal, Canada, May 15-16, 1956 (afternoons only).

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October 2-8, 1955.

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